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MEDICINE IN PEACE AND WAR.*

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It is not often, I fancy, that a philosopher and a humanist wholly ignorant of medicine is invited to address the graduating classes of a distinguished medical school. The very fact that I have been given this privilege betokens a clear realization on your part that the art of healing is, in its very essence, a humane enterprise which deeply concerns the common man because it contributes so greatly to his welfare and happiness. It is as a common man that I speak to you this evening—as a potential patient and as a representative of that large multitude of men, women, and children to whose service you are dedicating your lives.

In many respects the responsibilities which you, members of the graduating classes, are about to assume in a world at war are precisely the responsibilities which you would have had to assume in a world at peace. For yours is a profession dedicated to unremitting warfare against an enemy which never ceases its onslaughts. Disease signs no armistice, no peace treaty; it is a foe which never capitulates. You are all of you, each in his or her special field of medicine, signing up for the duration of a war that will outlast you and your successors through countless generations.

And yet, in other important respects, the present crisis concerns you as vitally as it does the rest of us. It concerns you professionally because of the wounds which your profession must attempt to heal, and also because war between man and man affords our common enemy, disease, a golden opportunity to diversify and redouble its attacks upon the human race. If medicine is indispensable in times of peace, it is doubly so in times of war.

But the present crisis concerns you also as human beings, as men and women who must keep the faith in a period of widespread skepticism and dismay. It is our lot, whether we like it or not, to be alive in an age of unprecedented social and spiritual upheaval. Seldom if ever have the very foundations of a culture been so shaken, the basis of ultimate belief rendered so insecure. Our nation, and we as individual citizens, are being challenged to declare the faith that is in us, to prove our metal, to demonstrate our nerve. What do we, individually and collectively, really believe in passionately enough to live for and, if necessary, to die for? What is our basic philosophy of life? What is the object of our highest allegiance? These are questions which each of us must face as honestly and intelligently as possible, because the course of events is already compelling us to answer them, and answer them without delay.

Medicine in a world at peace and medicine in a world at war—this is the dual topic which I invite you to consider on this solemn occasion.

I have been asking myself during the past weeks what it was that probably induced you to enter upon the training which you have just completed? Why have you chosen to become doctors and pharmacists, dentists and nurses? Can it have been primarily the hope of financial remuneration? Is money your chief concern—financial security, or economic comfort, or even wealth? To ignore or minimize the importance of what Plato calls "the art of wages" would be absurd. Even a philosopher need not be *that* unrealistic and other-worldly. The art of wages is an art which each of us must master if he is to live in a free society. And I, for one, believe that special talents and special industry merit unusual financial recognition. You are all entitled to financial security, and it is right and proper that each of you should

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receive an adequate economic reward for your services to your community.

But Plato, having duly recognized the art of wages, proceeds to make it clear that this art must, in any healthy society, be subordinated to those arts which are dedicated to more basic and more enduring human values. The true shepherd, he points out, devotes himself primarily to the welfare of his flock, the true carpenter, to the excellence of his craft, the true statesman, to the welfare of his people, the true doctor, to the health of his patients and not to his own financial gain. What Plato is here insisting on is the fact that all the serious professions in a well-regulated society are, each in its own way, a public trust, and that the truly professional man is one whose highest purpose is to contribute to human welfare. Only thus can man discharge his social responsibility and be a credit to his profession.

Plato also points out what is often forgotten, that only thus can we hope to achieve true happiness. This is one of the enduring paradoxes of human existence, that the surest way to achieve happiness is to ignore it and to devote one's self wholeheartedly to the task in hand, provided that this task has its own intrinsic interest and provided that it benefits mankind. This is especially true in the profession which you are entering. No profession has more intrinsic fascination; few are of greater value to man. If you prostitute your high calling by using it merely as a means to financial gains you will deserve the contempt of your fellows and inevitably forfeit your own self-respect. If, on the other hand, you regard your talents and your training as a public trust, a great adventure and a priceless opportunity to relieve human suffering, your reward will be the respect and love of all you serve. Sir William Osler once received a telegram which read, "Save my son George Halloran. God will pay all expenses." A doctor would certainly be ill-advised to rely too exclusively on God as his pay-master, but no doctor is worthy of the name who is not willing and eager to relieve suffering irrespective of financial remuneration.

If it is not mere financial gain, what is it, then, that has attracted you to medicine? There are, I believe, two sufficient reasons, and only two, for entering your profession—an interest in the science and art of medicine for its own sake, and an interest in human beings for their own sake. In some of you one of these twin motives may be predominant, in others, the other motive. But those of you who are destined to be truly successful in your noble calling are precisely those in whom both interests are strong and strongly interfused. Your interest in, and respect for, people will enable you to use the science of medicine wisely, and to remember always that it is a means to a human and humane end—the preservation and enrichment of human life. Your interest in the theories and techniques of medical science will enable you to practice the art of healing in all its several branches with ever increasing skill. If you stifle or neglect to cultivate either interest you will jeopardize and ultimately destroy your usefulness; you will become inhumane scientists or sentimental quacks. If you combine reason and emotion, knowledge and love, your contribution will be incalculably great.

The science of medicine depends today, as never before, upon effective cooperation among specialists and between the specialist and the general practitioner. The specialist and the diagnostician are both essential, and neither can function with maximum safety or efficiency save through the continued cooperation of the other.

Our scientific knowledge has increased so enormously that no human being can hope to master, in a single lifetime, all that is known concerning the body and its ailments. Specialization in every field of medicine is therefore imperative, if available knowledge is to be put to use and if new knowledge is to be achieved. Mere specialization, however, will not suffice, either at the practical or at the theoretical level. At the level of medical theory, the narrow specialist becomes increasingly unable to interpret his findings in the light of all relevant facts. He loses his perspective, and ends with a distorted apprehension of the whole situation. The part is taken for the whole, causes are mistaken for effects and effects for causes, until a just appreciation of the complete picture is finally lost. Just because the organic is conditioned by the inorganic, conscious states by unconscious processes, and, on occasion, bodily health by mental attitude, the several medical disciplines which you have been studying during the past years are themselves mutually dependent. Every specialist is therefore dependent on every other specialist; each must learn from the rest if he is really to understand man's incredibly complex psycho-physical nature.

Cooperation is equally essential at the practical

level. Here the specialist and the general practitioner must work hand in hand, since each has his peculiar limitations and his distinctive strength. The specialist has at his command a knowledge and a skill which the general diagnostician and practitioner cannot rival. But the latter, if he is as competent in his line as the specialist is in his, can envisage the total situation far better than any specialist, however distinguished, and can protect the patient against the dangers of a too limited and restricted diagnosis. The specialist sees, as it were, a horizontal cross-section of the case; he brings his knowledge to bear upon the patient's present condition, and concentrates upon one or another aspect of this present state. The able practitioner, in contrast, knows the case in vertical perspective; he is able to interpret present symptoms in the light of the patient's entire history and in terms of his total condition. The contributions of the specialist and the practitioner thus complement one another. The men to fear in medicine are, on the one hand, the specialists who ignore the work of other specialists and who scorn the wisdom of the general diagnostician, and, on the other hand, the practitioners who fear to confess their lack of specialized knowledge. The men to trust are those specialists and non-specialists who are most keenly aware of the complexities of medicine, of their own inevitable limitations, and of the imperative need for incessant cooperation. In medicine, as in government, your motto must be, *"E pluribus unum"*—"United we stand, divided we fall."

But the art of medicine requires more than mere scientific skill, however specialized or however synoptic. It depends also upon a recognition of human values and human personality. For patients are not mere "cases"; they are human beings who turn to you, doctors and nurses, for human understanding and sympathy. The science of medicine, like all science, is a body of knowledge and a technique of intellectual inquiry. As such, it is, by definition, impersonal, dispassionate, and objective. The art of medicine, in contrast, is the humane application of this knowledge to the alleviation of human suffering and the promotion of human welfare. The *art* of medicine is, therefore, essentially personal and human, not coldly impersonal or scientifically dispassionate. It is concerned with human individuals, human hopes and fears. The importance of this fact can hardly be exaggerated.

It is one of the tragedies of medicine today that the very complexities of modern science and of medical techniques tend so powerfully to accentuate the impersonality of medical treatment. Your great laboratories and gigantic hospitals, your complicated routines of consultation and diagnosis, the need for discipline, and the impersonal objectivity which necessarily characterizes all truly scientific inquiry, encourage a factory-like routine and a neglect of the human factor. But the patient, just because he is a human being, and because he comes to you in physical distress, craves not merely your scientific skill but your human understanding and sympathy. He is in trouble and afraid, afraid to hear your verdict, afraid to face whatever suffering may be in store for him. He wants to know the truth, however grim, yet fears the truth. He turns to you not only for your skill but also for friendliness and courage, sympathy and understanding. Can you remember this in the busy days and nights that lie ahead? Can you imaginatively identify yourselves with the patients, and, despite all routine and all weariness, respond to their human needs? Yours is a unique opportunity, as doctors and as nurses, to help your fellowmen in times of suffering and loneliness, anxiety and fear.

You will be able to measure up to this unparalleled responsibility only if you have within you great spiritual resources. The cure of bodies is an exhausting task. The comfort of the sick soul is far more exhausting and far more difficult. To fail here is excusable and tragic; to succeed here is to succeed indeed in the art of medicine. Here the essential requirement is a philosophy of life that you can live by and help others to live by. You need a religious anchorage so secure that you can preserve your own faith and courage in the face of suffering and death, and, in addition, instill your own assurance into the hearts and minds of those in pain and in the shadow of death. This will be the supreme task of your manhood and your womanhood, of your competence as human doctors and human nurses. If you fail here the world will still be grateful for your scientific and technical skill; but if you can combine this skill with love, courage and faith, you will be loved in return as it is given few men and women to be loved.

What I have said thus far concerns the art and science of medicine in time of peace. In time of war the dangers, the opportunities and the responsibilities of your profession are all increased—the dan-

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gers of too much and of too little specialization, the dangers of regimentation and callous impersonality, the opportunities for the exercise of technical skill and human understanding, and the responsibilities which all these opportunities imply. In war the doctor and the nurse are the great non-belligerents, the great humanitarians. Friend and foe suffer alike, and medicine ministers to all suffering because it is human suffering. Whatever the rights and wrongs, the justice and injustice, in any war, all are agreed on the supreme value and indubitable rightness of medical aid to the wounded and the diseased. The graduation exercises of a medical school are always an important occasion; in time of war their importance is momentous because they signalize the arrival of new reinforcements where reinforcements are so sorely needed.

But what of the large issues now at stake—the issues over which this war is being fought? My own convictions on this score are those of an ever increasing majority of my fellow citizens, those repeatedly voiced by the President of our United States. Democracy, we believe, must and will survive, because the values which we all cherish—freedom of thought and action, of intellectual inquiry and creative effort, of human dignity which makes slavery an outrage and a curse—because these values are so essentially a part of the democratic spirit and way of life.

Those of you who analyze the present situation in this way will do your part with courage and assurance. To those of you who still have doubts, let me suggest an application of the art of medicine to the wider arena of life. What does a doctor do in emergency? He decides and acts in the light of all available knowledge, even though he is perfectly aware that such knowledge is inconclusive and finite. No doctor ever acts on the basis of complete certainty; no doctor worthy of the name ignores such knowledge as is available. His action is always, of necessity, an act of reasonable faith. The belief of the quack is blind and credulous; the doctor who lacks all faith, all willingness to act in the absence of complete knowledge, will never act at all. The art of healing involves both faith and knowledge, as much knowledge as possible, and the courage to venture all on the basis of what is known.

This, I submit, is the procedure which all of us should follow in the present crisis. Many of the issues are indeed confused; our knowledge is indeed limited; the future is indeed uncertain. There are those who, as a result, counsel us to refrain from action, to wait, to watch, until Utopia is in sight. That way madness lies. It behooves us, in a time of crisis, to decide, as best we may, and to act boldly and resolutely in the light of this decision. Now is the time to summon up such faith and courage as is in us and to proceed, with a doctor's willingness to face all risks, along the road which lies ahead. You who are trained in the science and the art of medicine can provide us all with an example of reasoned assurance. I am confident that here too you will not fail us.

SPONTANEOUS HYPOGLYCEMIA IN CHILDHOOD.*

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In electing to describe our very limited experience with spontaneous hypoglycemia in children it is not with the intention of discussing a rare clinical state but rather because we believe the condition to be far more common than one is led to believe. Within the past two and one-half years we have had the opportunity to observe six such cases which will be described at this time. Our pediatric service is not a large one but our experience suggests that it is not a rare syndrome. Looking back over our cases we are confident that other fatal instances of this condition were not infrequently observed and were undiagnosed. No doubt others have had similar experience. The serious nature of such cases and the dramatic response to treatment in suitable instances, prompts us to make this report. Failure in the past to recognize and adequately treat severe and spontaneous hypoglycemia no doubt is the answer to some of the cases shown on hospital records as death from convulsions with cause unknown.

A discussion of this subject at this time seems

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peculiarly appropriate. We refer to the pioneer work and subsequent contributions of Dr. Seale Harris.

In reviewing the literature on spontaneous hypoglycemia in children one is impressed with the scant mention of it in the usual sources of pediatric reference, and it is, therefore, not surprising that cases of this nature continue to go undiagnosed.

In a partial review of American literature many excellent discussions pertaining to this subject are found. In a fairly comprehensive review of American literature we were able to find thirty-odd reports of spontaneous hypoglycemia of childhood exclusive of those occurring in newborn infants. In only one instance was the condition associated with adenoma of the pancreas.

In commenting on the probable mechanism of the hypoglycemia in the recorded cases the conclusions of the various authors were in many instances of a speculative character due to the mild nature of many of the recorded cases and consequent lack of postmortem material. Prominent among the suggested etiological factors are hyperactivity of pancreatic islets, insufficient carbohydrate intake, infection and liver damage, endocrine imbalance, and organic disease of the brain. Significant postmortem findings include hyperplasia of the pancreatic islets, liver damage and lack of adequate glycogen storage, hemorrhage into the adrenal glands (newborns) and central nervous system changes.

Especial attention should be called to the usual degree of hypoglycemia that occurs at times in premature infants and full-term infants, particularly in those born of diabetic mothers. We are unable to report a single case of this nature and our experience is no doubt hidden under such misnomers as intracranial hemorrhage, congenital atelectasis, prematurity and unexplained infections of the newly-born. While routine blood sugar determinations are not a part of our nursery routine it is our custom to start oral administration of five per cent glucose shortly after delivery and to continue its administration until breast feeding or artificial feeding has become well established.

Case No. 132944 (Case I).—This three-year-old white child was admitted to the University Hospital July 8, 1937, with the complaint of convulsions and high fever of four hours' duration. Thirteen hours before admission she became quite fretful but no other unusual abnormalities were noted. Late on the morning of admission she became worse and had a diarrheal stool. The mother thought the child was feverish. One hour later she again passed a diarrheal stool. The diarrhea continued until admission to the hospital. In the early afternoon of the same day the child had a convulsion which lasted for ten minutes, followed by coma. A local physician was called and on arrival found the child in another convulsion. Morphine and phenobarbital were administered. At this time the temperature was 105 degrees rectal. While on the way to the hospital the child had numerous slight convulsions and several generalized convulsions.

Physical examination revealed a well developed child of four years of age in a comatose condition. The skin was pale, hot and moist. Respirations were fast and jerky. Mucous membranes were pale. The sclerae were clear. Pupils were dilated and fixed. Examination of the lungs was negative except for large rhonchi. The pulse rate was 176. The usual reflexes were not elicited.

Lumbar puncture showed the fluid to be under no increase in pressure and the fluid was clear and colorless. Spinal fluid protein was not increased. There was an atypical reduction in Fehling's solution with eight drops of spinal fluid. The cell count was 2. The urine showed 2+ albumin, 4+ acetone, eight to ten white blood cells, an occasional red blood cell and granular casts. The carbon dioxide combining power was 35 volumes per cent and the blood sugar (venous) was so low that it was impossible to read it with ordinary standards.

Attempts were made to control the convulsions by magnesium sulphate intravenously and ether inhalations. Before glucose infusions could be given the child died.

The significant autopsy findings were as follows: *Adrenals*—Hemorrhage into both adrenal glands involving more of the cortex than the medulla, the left adrenal more than the right. The structure of each adrenal was not completely destroyed. *Liver*—Cloudy swelling. The liver cells contained an unusual amount of fat. *Pancreas*—No change noted.

It is reasonable to suppose that hemorrhage into the adrenal glands was the immediate cause of the hypoglycemia, but why the hemorrhage? The febrile course suggested unexplained infection. Hemorrhage into the adrenal glands of the newborn associated with hypoglycemia is not an unusual finding, but must be a very infrequent cause of hypoglycemia of older infants and children.

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Case No. 140966 (Case II).—A seven-year-old white male was admitted to the University Hospital July 3, 1938. He was apparently in good health until four and one-half hours before admission at which time the mother heard the patient groaning and went in to see about him. At this time he complained that he could not stand and very quickly lapsed into unconsciousness. Clonic convulsive movements were noted, most marked on left side.

Physical examination on admission revealed a moderately well developed and nourished white male of seven years of age. He was comatose and having clonic convulsions mainly involving the left side. The skin was hot and dry. The mucous membranes were slightly cyanotic. The sclerae were moderately injected. The pupils were widely dilated, equal, and reacted very sluggishly to light. There were coarse nystagmoid movements to the left. The heart rate was rapid. The blood pressure was 148 over 80. The temperature was 104.8 degrees rectal. Respirations were labored and deep. Auscultation revealed a few coarse rhonchi over both lungs. The left leg and left arm exhibited marked clonic convulsive movements. The right arm and leg were moderately flaccid. The usual reflexes were present bilaterally but were markedly weaker on the left.

Lumbar puncture showed a clear fluid with no increase in pressure. There was no reduction with 2cc. of fluid in Fehling's solution. The cell count was one lymphocyte. Carton dioxide combining power was 30 volumes per cent. The blood sugar (venous) was 24 mg. per 100 cc. of blood.

The convulsions were controlled with ether inhalations and small doses of magnesium sulphate hypodermically. In spite of infusions of glucose and Hartman's solution intravenously the patient remained in a comatose state and death occurred twenty hours after admission. Permission for autopsy was not granted.

The cause of the hypoglycemic state in this instance was not ascertained. The febrile state of the patient suggests that an undiagnosed infection may have been the essential etiological factor. In fact, it is not at all certain that hypoglycemia was directly concerned with the fatal outcome but rather a part of the moribund state. It is interesting to note that convulsions ceased after administration of glucose and sedatives, yet the patient remained in a comatose state.

Other references are found in the literature in which patients with exceedingly low amounts of blood sugar continued to show symptoms referable to the central nervous system after adequate treatment with intravenous glucose. It is conceivable that excessive amounts of insulin acting over a period of hours may cause lasting damage to the central nervous system which cannot be corrected by the usual glucose therapy.

Case No. 141147 (Case III).—This two and one-half year old white female was admitted to the University Hospital July 10, 1938. She was apparently in good health until twelve o'clock noon, twelve hours before admission. At this time she became very irritable and vomited several times. She refused food but took fluids fairly well. In about four hours she became maniacal, would not walk and did not recognize her parents. She seemed feverish and pulled at her left ear. There were occasional twitchings of the extremities but no generalized convulsions. Food was not taken for thirteen and one-half hours before admission. About seven hours before admission her breathing became labored and deep. These symptoms increased gradually until admission. She became more violent and was seen by her local physician who gave her a hypodermic and recommended hospitalization.

Physical examination on admission revealed an acutely ill two and one-half year old white female. She was in a semicomatose state. Sweating was pronounced and respirations were deep and labored. Her mucous membranes were pale. The skin of the neck and upper frontal region showed moderate brownish pigmentation. Eyes showed converging strabismus and the right pupil was irregular. Both pupils reacted sluggishly to light and the corneal reflex was depressed. The fundi showed an engorgement of the veins. A few rhonchi were heard over both lung fields. Heart rate was fast and of poor quality. The temperature was 99.2 rectal. The respirations were 56. Both arms and legs showed coarse clonic movements. Knee and ankle jerks were equal and hyperactive. The Babinski reflex was questionably positive, more marked on the right. Kernig was negative.

Lumbar puncture revealed the fluid to be under no increased pressure, clear, and colorless with slightly increased protein. There was a reduction with 12 drops in Fehling's solution. Cell count was zero. Blood culture was negative. Blood sugar (venous) was 19 mg. per 100 cc., and carbon dioxide combin-

ing power was 21 volumes per cent.

The patient was given soda bicarbonate intravenously and this was immediately followed by 25 gm. glucose solution intravenously. Following this the patient had no more periods of apnea which had previously occurred about every thirty seconds. In addition she was given adrenalin which gave her temporary relief but she continued to have convulsions which gradually became more severe. The temperature gradually rose from 99.2 to 102.6 with pulse rate of 208. Glucose infusion was again given after five hours. The patient remained in a semicomatose condition and death occurred about eight hours after admission. Permission for autopsy was not granted.

Again we are unable to explain the cause of the existing hypoglycemia and to advance a satisfactory reason as to why glucose therapy was not effective.

Case No. 146445 (Case IV).—This twenty-two months-old white male infant was admitted to the University Hospital October 19, 1939, with the history of vomiting of eighteen hours' duration. Shortly before admission he became drowsy and could not be aroused. No history of infection was obtained. Past history was of particular importance in that he was seen at the University Hospital a few hours after birth with all the earmarks of intracranial hemorrhage. The child has never sat alone, walked or talked, and at times convulsive moments of both extremities have been noted.

Physical examination showed a well developed infant in a markedly comatose state. The temperature was 98 degrees rectal. The respirations were slow and deep in character. There were occasional twitchings of all four extremities and dorsiflexion of both feet. All extremities were somewhat rigid. The deep reflexes were sluggish. The pupils were equal and reacted normally to light. There was slight diverging strabismus of both eyes. The general appearance was that of a moribund infant.

Laboratory findings were as follows: Hemoglobin 84 per cent, Rbc 5,180,000 and Wbc 7,700. Blood smear was essentially normal. The Wassermann test was negative. Urinalysis twelve hours after admission showed 4+ acetone. Blood sugar (venous) on admission was 21 mg. per 100 cc. Carbon dioxide combining power was 36.9 volumes per cent.

Treatment consisted of 10 cc. of 50 per cent glucose intravenously, 5 m. of adrenalin and 5 per cent glucose given by intravenous drip. In addition 450 cc. of one-sixth molar sodium lactate was given intraperitoneally. Improvement in the child's general condition was prompt although he continued to vomit for about eight hours. Eight hours after admission blood sugar (venous) was found to be 189 mg. per 100 cc. Six days after admission the child was discharged from the hospital in good condition.

Seven months later he was brought back to the hospital with a similar attack. The history at this admission was as follows: He appeared well until twelve hours before admission at which time he became restless and extremely thirsty. Three hours before admission it was noticed that he appeared drowsy and could be aroused only with difficulty. Several small convulsive seizures were observed by the mother. On admission to the hospital he appeared pale and was in a comatose state. Respirations were slow and deep. The usual reflexes were present but sluggish. Dorsiflexion of both feet was again observed. Temperature on admission was 96.4 degrees rectal. Again the general appearance was that of a moribund infant.

Laboratory examination was as follows: Hemoglobin was 81 per cent, Rbc 3,780,000 and Wbc 8,120. Blood smear was essentially negative. Urinalysis was normal. Blood sugar (venous) on admission was 26 mg. per 100 cc. and the carbon dioxide combining power was 37.6 volumes per cent.

Treatment consisted of 20 cc. of 25 per cent glucose intravenously and 5 per cent glucose by intravenous drip. Immediately following the administration of glucose the child's color appeared better and drowsiness disappeared. One hour after initial glucose, blood sugar (capillary blood-micro method) was found to be 261 mg. per 100 cc. Five and one-half hours later the blood sugar was 135 mg. per 100 cc. and the patient appeared to have completely recovered. Five days after admission the patient was discharged from the hospital in good condition.

Two weeks after discharge from the hospital he was returned for interval examination. At this time blood sugar (capillary blood-micro method) was 107 mg. per 100 cc. and the mother stated that the child had appeared in good health.

One week later he again was brought to the hospital with a history of having been well up until one-half hour before admission. Mother states he appeared pale and that his eyes rolled back and that she feared another "spell". She promptly gave the infant two tablespoons of Karo corn syrup. This was followed by immediate improvement. On question-

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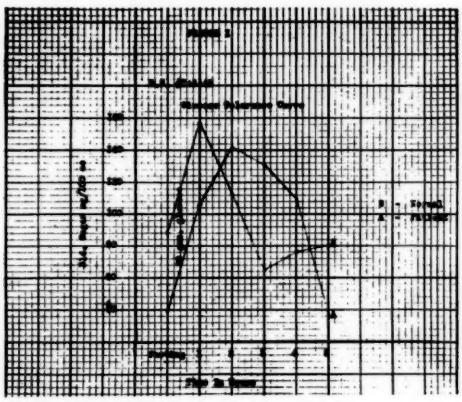
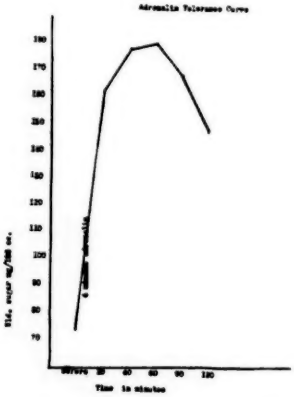
ing it was found that she had failed to give the usual sweetened orange juice. Blood sugar (capillary blood-micro method) at this time was 280 mg. per 100 cc.

The glucose tolerance curve at the time of an interval examination is shown in Figure I. The fasting level and five hour determination is compatible with this patient's hypoglycemia state.



Adrenalin response curve at the same interval examination shown in Figure II suggests no interference with glycogen mobilization during a symptom-free interval.



The recurring hypoglycemia state of this infant may perhaps be attributed to intracranial injury. Similar reports are not infrequent in the literature on this subject.

Case No. 158065 (Case V).—A three-year-old white male was admitted to the University Hospital May 15, 1940, with the history of having been in good health up until 6 A. M.—four hours before admission. At this time he awoke, vomited and had a generalized convulsion. Vomiting and convulsions persisted at frequent intervals up until the time of admission. In the meantime he was seen by a local physician who made a diagnosis of pin worms and prescribed for these. The child continued to have convulsions and consequently was brought to the Hospital.

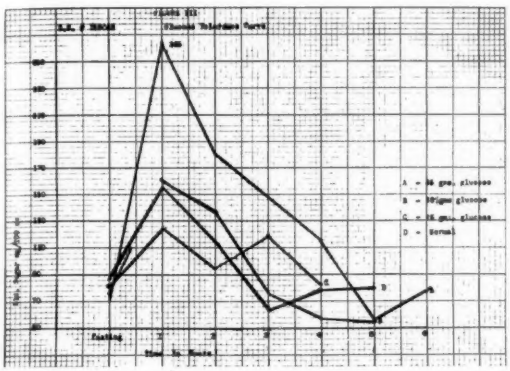
Physical examination on admission revealed a well developed and nourished three-year-old white boy with generalized convulsions and opisthotonos. Eyes were open and staring. There was foaming at the mouth. The skin was dry and doughy. The mucous membranes were markedly pale. Examination of the eyes showed the lids open, pupils dilated and fixed and reacting only very slightly to light. The corneal reflexes were absent. Both eardrums were dull and slightly injected. The upper front incisors were decayed and only the stumps were present. The throat was injected and the tonsils were enlarged and injected. The anterior cervical glands were enlarged. The respirations were slow and deep and coarse rhonchi were heard over both lung fields. All of the extremities were held in a rigid position and the knee jerks were hyperactive and equal. There was an unsustained bilateral ankle clonus. The temperature was 99.6 degrees rectal.

Blood examination revealed the hemoglobin to be 55 per cent, Rbc 4,160,000 and Wbc 9,200. Smear showed a shift to the left. Urine showed a 4+ acetone but was otherwise negative. Blood sugar (venous) was 35 mg. per 100 cc. and carbon dioxide combining power was 35 volumes per cent.

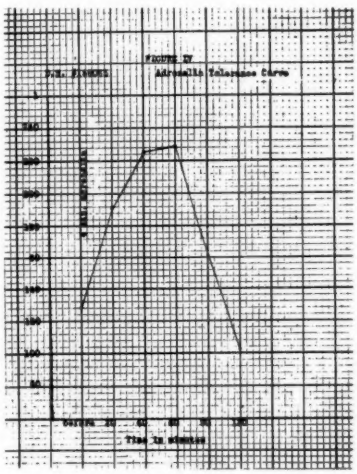
The patient was immediately given 4 min. adrenalin and 20 cc. of 25 per cent glucose intravenously. After about ten minutes the patient began to arouse and asked for water. After about twenty minutes he was perfectly rational and appeared normal in every respect. Intravenous drip of 5 per cent glucose was started and one-sixth sodium lactate solution was also administered. One and one-half hours after the administration of the initial dose of glucose the blood sugar was 224 mg. per 100 cc. of blood. After several hours the patient dropped into a rather heavy sleep from which he could be aroused and appeared rational when aroused. Vomiting stopped several hours after the blood sugar had become elevated.

Six hours after administration the blood sugar was 74 mg. per 100 cc. of blood and the following morn-

ing it was 85 mg. per 100 cc. of blood. After twenty-four hours the Murphy drip was discontinued and the patient was put on regular diet and orange juice, oz. 6, q. 4 hours. Subsequent interval glucose tolerance curves (capillary blood-micro method) after administration of glucose are shown in Figure III. Adrenalin response is shown in Figure IV.



The response to intravenous glucose would seem sufficient proof to justify the conclusion that the symptoms present in this case were directly due to hypoglycemia. To date the child had not had further attacks and it is impossible to state where the breakdown in carbohydrate metabolism occurred.



Case No. 159232 (*Case VI*).—A four-year-old white male was admitted to the University Hospital June 28, 1940, with the history of having drunk about one-fourth pint of whiskey thirty-six hours before admission and having remained unconscious since that time. There was no vomiting or convulsions. The temperature was 100.6 degrees rectal.

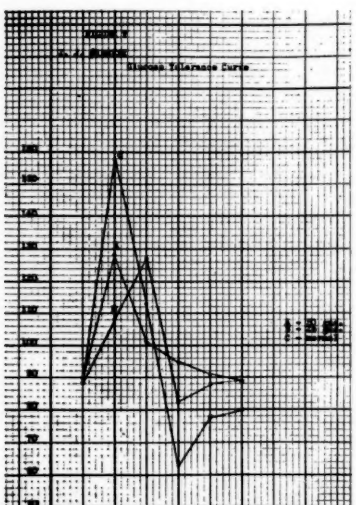
Physical examination on admission revealed a well developed and nourished four-year-old white male. He was unconscious, having loud noisy respirations, was slightly cyanotic and difficult to arouse. The skin was hot and dry. The mucous membranes were moderately cyanosed. Pupils were alternately dilated and constricted and reacted sluggishly to light. Sclerae and conjunctivae were slightly injected. Fundi were not visualized. Tongue showed a heavy whitish coating. Throat and tonsils were mildly injected. There were numerous large rales over both lung fields. The abdomen was scaphoid. The skin was somewhat doughy and of poor turgor. The liver edge was barely palpable. There was a small swelling about 2 cm. in diameter in the subcutaneous tissue at the middle of the mandible. There was a mild erythema of the skin over this area. There was soft tissue swelling about 3 cm. in diameter over the left hip. The skin was red and hot over this area. All reflexes were sluggish. A diagnosis of acute alcoholism, acidosis and hypoglycemia was made. Blood sugar (venous) was 28 mg. per 100 cc. Carbon dioxide combining power was 25.3 volumes per cent. Spinal fluid was negative.

After admission slight twitching of the hands and feet were noted. There were no signs of generalized convulsions. 960 cc. of one-sixth molar sodium lactate solution was given intravenously. At this time the report of the blood sugar was obtained and 20 cc. of 25 per cent glucose was given intravenously. About two minutes following this the patient roused and began asking for food and water. 2.5 per cent glucose intravenously was started at a slow rate. One and one-half hours after initial adminstration of glucose the blood sugar (venous) was 282 mg. per 100 cc. At this time the patient was rational and recognized his parents. It was noticed several hours after admission that there was a slight ptosis of the left lid, dilatation of the left pupil and weakness of the left medial rectus muscle. This was thought to be the result of a peripheral third nerve neuritis probably due to the alcohol. About six hours after admission the patient became markedly hyper-irritable with involuntary jerky movements at the slightest stimulant.

The patient was put on orange juice, oz. 8, with Karo every four hours and compresses were applied to the soft tissue swelling. The following morning

he appeared very drowsy and was hard to arouse. The blood sugar (venous) at this time was 58 mg. per 100 cc. Orange juice with Karo was gavaged and two and one-half hours later blood sugar (venous) was 245 mg. per 100 cc. The following morning blood sugar (venous) was 63.5 mg. per 100 cc. On the third day the temperature was 104.6 degrees rectal and examination revealed many fine rales over both lung fields. X-ray of the chest revealed a diffuse bronchitis. Wbc were 10,000 and blood sugar (venous) was 63.5 mg. per 100 cc. The patient was started on sulfanilamide therapy. The temperature continued to remain elevated, gradually returning to normal on the tenth day and remained so throughout the remainder of his stay. The peripheral neuritis gradually cleared up. Gavage had to be resorted to up until the ninth day at which time the tube was removed and from then on the patient took his feedings voluntarily. The bronchitis gradually cleared up and after the tenth day the patient appeared perfectly rational and wide-awake. The soft tissue swelling gradually subsided.

Glucose tolerance curve done on two occasions was as follows:



The curve representing the levels of sugar in the blood may be considered low when the sugar in the blood does not rise more than 40 mg. per cent above the fasting level. The type of curve in this instance is not conclusive of hypoglycemia and of no material aid in diagnosis.

The prompt response to intravenous glucose would appear to justify the conclusion that coma in this instance was the result of hypoglycemia and not due to the depressing effect of large amounts of alcohol. We know of no reports in medical literature in which acute alcoholism was associated with hypoglycemia. We are, however, of the opinion that alcohol was the immediate cause of the hypoglycemia noted in this instance. In support of this suggestion we record the following case inadequately studied from the standpoint of hypoglycemia. The similarity between the two cases is striking and the fact that the single recorded blood sugar determination was considerably below the usual normal reading even after glucose administration suggests that a very real hypoglycemic state may have been present.

A five-year-old colored female was admitted to the University of Virginia Hospital May 6, 1940, with a history of having been in good health up until thirty-six hours before admission. At this time she drank an unknown quantity of "moonshine" whiskey. About an hour following this the patient vomited, became irrational and violent and lapsed into coma at times. A short while later she began having generalized convulsions which lasted only for a short period but were repeated frequently. No food or water was taken from time of drinking whiskey up until admission. On admission the temperature was 99.4 degrees rectal.

Physical examination revealed a well developed but poorly nourished five-year-old colored female comatose but violent and irrational when aroused. Skin was dry with loss of turgor. Neck was voluntarily held stiff. Other than this physical examination was essentially normal. Urinalysis showed 3+ acetone, otherwise normal. Carbon dioxide combining power was 34.8 volumes per cent.

Patient was given 920 cc. of one-sixth molar sodium lactate solution intravenously. During the first day of hospitalization the patient remained comatose and was aroused only with difficulty. 750 cc. of five per cent glucose in normal saline was given intravenously on the morning following admission. Following this infusion blood sugar was 64 mg. per 100 cc. Patient continued somewhat drowsy during the second day. On the third day she became more responsive and began taking foods and fluids. She was discharged on the fifth hospital day in good condition (Case No. 110032).

We have attempted to produce hypoglycemia in

puppies by the administration of alcohol: Experiment I—To four of five puppies weighing approximately 750 gms. 25 per cent ethyl alcohol was administered by gavage in quantities sufficient to produce inability to walk or stand. Blood sugar determinations were made immediately before administration of alcohol and 48 hours after. No food was given between this period (see Chart I). Experiment II—Seventeen hour fasting blood sugar was determined. No food was given; alcohol given to three of five puppies on two occasions five hours apart in quantities described above. No food was given for a period of sixty-five hours or forty-eight hours after initial administration of alcohol. Blood sugar were determined at this time (see Chart I).

This procedure failed to produce in puppies a hypoglycemia which could not be explained by the prolonged fast.

CHART I

BLOOD SUGAR DETERMINATION ON PUPPIES AFTER FASTING AND INGESTION OF ALCOHOL

EXPERIMENT I

Puppies	1 hr. after feeding	Alcohol given	24 hr. fasting and 24 hrs. after alcohol	48 hr. fasting and 48 hrs. after alcohol
1	143 mg./100 cc.	Control	125 mg./100 cc.	77 mg./100 cc.
2	133	Treated	118	59
3	138	Treated	118	59
4	154	Treated	118	67
5	133	Treated	125	69

EXPERIMENT II

Puppies	17 hr. fasting	Alcohol	48 hrs. after alcohol 65 hrs. fasting
1	105 mg./100 cc.	Control	61 mg./100 cc.
2	105	Treated	65
3	100	Treated	55
4	100	Treated	53
5	91	Control	63

Table No. I lists the symptoms and signs arranged in order of their frequency:

TABLE I

Hypoglycemia	7
Acidosis	7
Coma	7
Convulsions	6
Sudden onset	6
Sluggish reaction of pupils to light	6
Cyanosis	4
Negative spinal fluid	4 (3 not done)
Vomiting	3
Elevated temperature	3
Dilated pupils	3
Slow and deep respirations	3
Labored and deep respirations	2
Jerky and deep respirations	2
History and signs of birth injury	2
Evidence of infection	2
Prolonged fasting period	0

RESPONSE TO GLUCOSE

Immediate and marked	4
Slight	1
None	1
Glucose not given	1

The diagnosis of severe hypoglycemia is not difficult and can be made with surprising accuracy from the symptoms and signs alone even by those of limited experience. Failure to recognize such cases is the result of unfamiliarity with the clinical picture rather than to difficult details of diagnosis. It has been our experience that once an interne has had the opportunity to observe a severe case of hypoglycemia he is not apt to fail to make a proper diagnosis when the opportunity again presents itself. In every instance, however, the diagnosis must be verified, if possible, by fasting blood sugar determinations, five to six hour glucose tolerance curves and insulin tolerance curves. Since our very limited experience includes, with a single exception, cases presenting themselves in a first and severe attack we have not had any personal experience with the insulin tolerance test.

The value of the five hour glucose tolerance curve in the diagnosis of spontaneous hypoglycemia may well be questioned and certainly it was of no great aid in the diagnosis of the cases here reported. It must be admitted that the normal limits for the five hour curve have not been well established. In Figure VI (courtesy of Dr. H. B. Mulholland) the five hour blood sugar levels in five apparently healthy medical students are described. The marked variation in the five curves is obvious.

Hart and Lisa* found that hypoglycemia (blood sugar below 80 mg.) occurred in 2,371 of 21,000 patients whose blood sugars were taken on a fasting stomach. Of these 751 fell below 70 mg. A few blood sugar values were as low as 28 mg. without shock. They suggest that normal individuals may have

*_Endocrinology_, 27:1-160, July, 1940. Rate of Occurrence of Hypoglycemia: Study of 21,000 Routine Fasting Blood Sugars. J. F. Hart and J. R. Lisa, New York, p. 19.

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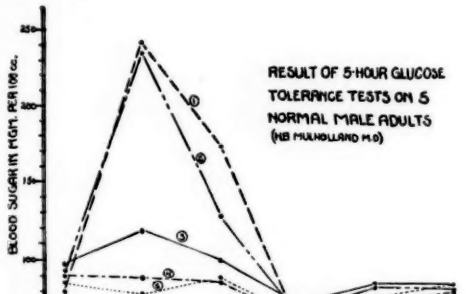
occasional low blood sugar values with an otherwise normal blood sugar, and conclude that spontaneous hypoglycemia is not a common condition, or routine fasting blood sugars are not reliable guides to its occurrence.


RESULT OF 5-HOUR GLUCOSE TOLERANCE TESTS ON 5 NORMAL MALE ADULTS
(NB MULHOLLAND M.D)
BLOOD SUGAR IN MGM. PER 100 cc.
100 grms. GLUCOSE
TIME

One cannot afford to fail to make blood sugar determinations in any case of unexplained unconsciousness and persistent convulsions. Failure to promptly make such studies is certain to result in loss of life.

Far too frequently one can only speculate as to where the breakdown in carbohydrate metabolism occurs. It is reasonable to suppose that in many instances more than one etiological factor is operating to cause an existing hypoglycemia.

TREATMENT

There is a general uniformity of opinion concerning the treatment of severe spontaneous hypoglycemia. Treatment consists of oral and parenteral administration of glucose and in those cases where proper mobilization of glycogen is not occurring adrenalin is a valuable adjunct in therapy although its effects are only temporary. Where response to therapy occurs it is usually prompt and gratifying, the patient frequently being restored to consciousness within a few minutes' time. Not all cases, however, have this pleasant outcome as illustrated by the fatal cases here reported. The mere finding of hypoglycemia does not mean that it is necessarily the cause of the presenting symptoms.

There is less uniformity of opinion concerning the treatment of less severe and repeatedly occurring hypoglycemia. Certain cases obtain relief by merely increasing the intervals of feeding, others by supplementary feeding of small amounts of sugar. In true insulin sensitivity good results have been reported from the use of low carbohydrate-high fat diets. The occurrence of pancreatic tumor necessitating operation is fortunately rare in children. Hartmann† suggests that before subjecting children to such a procedure the following criteria be established: (1) "There should be proof of more or less persistent or frequently recurring hypoglycemia. (2) It should be shown that the symptoms expected to be relieved by surgery can be relieved by dextrose administration intravenously. (3) It should also be shown that the symptoms cannot be relieved by conservative measures of diet regulation (4) In the case of recurrent attacks of hypoglycemia with normal intervals between attacks, it should be shown that one is dealing with insulin intolerance or hypersensitive type."

SUMMARY

Six cases of spontaneous hypoglycemia are reported. The fact that two of the cases here reported died without showing the usual response to therapy serves to emphasize the occasional serious nature of the condition. This fact has not been given sufficient mention in the usual sources of pediatric reference and the suggestion is therefore made that such cases occur with greater frequency than is usually believed.

Attention is called to an apparent relationship between acute alcohol poisoning and hypoglycemia in one instance.

NOTE—Since this article was submitted for publication we have observed two additional fatal cases in which there was marked hypoglycemia. Postmortem findings in the one case which came to autopsy were: hyperplasia of islets of Langerhans, dehydration, slight atelectasis—bilateral and hyperplasia of lymphoid structures.

†Hartman and Jaudon, 1937—*J. Ped.*, 11:1-36—July, 1937.

THE EYE FINDINGS IN DIABETES.*

WILLIAM WICKHAM TAYLOR, M.D.,
Norfolk, Virginia.

I have nothing original to offer you today, but I think it might be interesting to review the eye changes in diabetes. This disease is of interest to us all, and its eye complications are rather frequent and very important. Often the eye complications constitute the first complaint of a diabetic patient; and it is through the recognition of their true nature that the presence of diabetes is first suspected. In preparing this material I was struck by the fact that the same question comes up with regard to almost every one of the complications: namely, whether the complication is caused directly by the diabetes itself by some toxin or some derangement of metabolism, or, on the other hand, is due only indirectly to diabetes which reduces resistance in general and allows some other direct cause to take hold.

Because of its high frequency and its damaging nature, the most important eye complication is that of the retinal lesions seen in diabetic patients—the so-called "diabetic retinopathy". Here the question of the direct or indirect effects of the diabetes comes up with the greatest force. There is not time today to go into the arguments used, but suffice it to say there is, no doubt, much truth on either side. It is generally agreed that vascular damage can be found in the retinal vessels, if diligently enough sought for, in almost all cases of retinopathy associated with diabetes; and, further, that all sorts of senile and arteriosclerotic lesions are found in diabetes. But, clinically, there is one peculiar form of retinal lesion seen much more frequently in diabetic patients with arteriosclerosis than in non-diabetic patients with arteriosclerosis. It is necessary, then, to assume some connection between the diabetes and the type and distribution of arteriosclerotic lesion that accompanies it. This peculiar form of retinal lesion has come to be known as "diabetic retinopathy". Hirschberg, in 1890, first described the retinal picture seen in diabetes and gave it the name retinitis punctata centralis. Here hemorrhages are an extremely common finding. These are usually small, deep-seated, and are situated away from the large vessels, indicating that seepage through diseased vessel walls is the explanation for their formation. Where sepsis is present elsewhere in the body, in addition to the diabetes, extensive pre-retinal hemorrhages, or hemorrhages into the vitreous, may occur. These latter, strangely enough, usually occur where the diabetes is well-controlled. Needless to say they offer extreme prognosis, as eyes are usually lost where such hemorrhages take place. The exudates of diabetic retinopathy have a fairly characteristic appearance. They are small and circumscribed, and are shiny white in appearance with well-defined edges. Usually they occur in the central area, and often form a rough ring about the macula. This is quite different and easily distinguished from the well-known "macular star" of renal retinopathy. Like the hemorrhages, the exudates are inert and remain for long periods without undergoing visible change. Retinal edema is extremely rare in this condition, and retinal detachments do not occur. Pathological examination reveals no distinguishing features. Figures vary from 2 to 30 per cent as to the incidence of this complication in diabetic patients: a mean value of about 14 per cent being nearest correct. It is seen most frequently in old females and is very rare under the age of forty. There seems to be no relation between the occurrence and degree of the retinal lesions and the duration and severity of the diabetes, except that retinal lesions are rarely seen where the diabetic condition has not been present for at least three years. The symptoms depend on the site of the lesions. Where the macular is uninvolved by hemorrhages and exudates the vision is not impaired, and the lesions may be present for long periods without causing trouble; whereas, as soon as the macula is involved, the central vision is acutely reduced. Elimination of sepsis and careful control of the diabetes is essential. The presence of retinal lesions of this type does not entail the terrifying prognosis of renal retinopathy with regard to early death, for patients have been followed eight to ten years with it. But as a general rule prognosis as regard to vision is not good, for the changes tend to be progressive.

There is one retinal picture which is very characteristic of diabetes—that of lipaemia retinalis.

*Read before the Annual Clinic of the Norfolk County Medical Society, at Norfolk, Va., April 17, 1941.

Lipaemia is a condition in which the fat content of the blood is so greatly increased that it changes the appearance of the blood. It is present in a great number of conditions, such as diabetes, alcoholism, starvation, asphyxia, phosphorus poisoning, pneumonia, peritonitis, trench nephritis, and xanthomatosis. But, with the rare exception of leukaemia which has been treated with radiation, diabetes is the only one of the above diseases in which the condition becomes marked enough to be seen with the ophthalmoscope in the retinal vessels. This is the rarest eye complication of diabetes. It was first described in 1880 and since then only about fifty cases have appeared in the literature, 80 per cent of these occurring in females. It is seen usually in young people, being a rarity over the age of forty, and whenever seen the patient is approaching a state of coma. Clinically, the picture is unmistakable. The color of the fundus vessels becomes milky, first in the periphery, and this change progresses toward the disc. In the advanced stage the vessels become enlarged, flat, and ribbon-shaped, and they appear to be filled with milk. In some cases they have a yellow color and frequently there are yellow stripes along the vessels. The fundus takes a lighter color than normal, due to similar changes in the widespread vascular bed of the underlying choroid. The disc and fundi are otherwise normal. The retinal tissues are entirely normal in pathological section, except that the blood plasma in the retinal vessels is loaded with emulsified fat. This seeps into the perivascular spaces, thus giving rise to the yellow stripes mentioned above. Where this condition is found the patient is always very ill; though with the use of insulin the prognosis is now no worse than other cases of impending diabetic coma, whereas, before insulin was used, seventeen of the eighteen reported cases died suddenly of coma.

Transient blurring of vision in diabetic patients has long been known, but its true nature was not understood until Duke-Elder studied this subject in 1925 and stated that the refractive power of the lens varies directly as to the sugar content of the blood. That is, there is a tendency to hyperopia with decreased blood sugar, and, conversely, to myopia with increased blood sugar. This hypothesis has been well borne out in experience. Duke-Elder gave as his theory of the mechanism of this change in the refractive power of the lens, a change in the osmotic pressure of the aqueous concurrent with changes in the blood sugar level, resulting in absorption of fluid by the lens with swelling and myopia in hyperglycemia; and the reverse process resulting in hyperopia with hypoglycemia. This complication has been reported as the first complaint of diabetic patients in as high as 34 per cent in one series. So the possible presence of diabetes must be kept in mind in all cases of sudden changes in refraction, and it is important not to prescribe glasses for these cases until the diabetic condition is well regulated, for only then will the refractive state become fixed and normal again.

It is known that diabetic patients are prone to develop cataracts. We find two separate types of cataract in diabetic patients. The first are in no way different from the ordinary senile cataract, except that they develop more frequently and at an earlier age in the diabetic than in the non-diabetic patient. According to Friedenwald, this is explained by virtue of the prematurity of diabetics in general. The second type of cataract is the true diabetic cataract. These occur in young patients, are associated with severe forms of diabetes, are bilateral, mature rapidly and have the characteristic subcapsular changes of secondary cataracts. At the onset one sees many vacuoles just beneath the capsule of the lens, with flaky opacities appearing between them. Then extensive hydration of the underlying cortex takes place, as is shown by water clefts and separation of the sutures. This goes on to a general swelling of the lens fibers until the lens is uniformly cloudy and opaque. There are many theories as to the cause underlying these changes. Some of these are: a degenerated state of the ciliary epithelium affecting the nutrition of the lens; toxic products of metabolism in diabetes; endocrine failure; and the photo-chemical theory. But, by far the most attractive theory, to my mind, is that of hydration of the lens secondary to change in the osmotic pressure in the aqueous. It is easy to conceive of this cataract formation as an end stage of the swelling of the lens in the myopic refractive changes described above. In very early stages of this hydration, adequate control of the diabetic condition may occasionally arrest the process and even result in disappearance of opacities. But, generally speaking, once the opacities have begun to form the process is rapid, and a mature cataract soon results. Here surgery is the only form of treatment. Although this does not entail a bad prognosis provided the diabetes is well-con-

trolled, it must be stated that cataract extraction in diabetic patients does provide greater anxiety than those in non-diabetic patients. The two most dreaded complications are occurrence of hemorrhage and post-operative infection. Every care must be taken to avoid these. Systematic and close search must be made for the presence of foci of infection, and hypertension must receive special care here. Having taken these precautions, one may expect good results from operation, provided, further, of course, there is no pre-existent damage to the back of the eye. The patient should be under the close care of an internist for several months prior to operation to assure proper control of the diabetic condition during that period, and dietary and insulin dosage should be regulated in the hospital where the operation is to be performed several days before it takes place. Here, again, the use of insulin has greatly changed the prognosis. With insulin and the care outlined above these patients enjoy the same good prognosis of other non-diabetic cataract patients, while before the use of insulin 30 to 40 per cent of these patients lost their eyes from infection following operation.

There are several very rare eye complications of diabetes about which I will say only a few words. First, I want to mention the changes in the uveal tract. In the older literature diabetic iritis is a frequent finding, but such an entity has almost disappeared. Today it is pretty generally agreed that the rather frequent occurrence of iritis in diabetic patients is very definitely a manifestation of the general low resistance to infection so characteristic of this disease. However, there is a change in the iris which is very characteristic of diabetes. This takes place in the pigment epithelium and consists of accumulation of fluid under this layer, separating it from the underlying non-pigmented epithelium and forming curious blisters or cysts in this area. The pigment granules may be liberated from the swollen cells and become deposited on the anterior surface of the iris and the lens. This may be recognized clinically with the aid of the slit lamp in a few cases, while it is pathognomonic and easily picked up in pathological section. Still another condition in this group is "rubeosis of the iris". This is a rare condition in which the iris of diabetic patients shows a peculiar non-inflammatory proliferation of vessels. Rubeosis is usually associated with glaucoma of an intractable nature.

Optic neuritis and ocular palsies in diabetic patients are thought to be part of the general peripheral neuritis so often seen in diabetes. Here, again, there is great argument as to whether this is due to diabetes *per se* or only indirectly by the general lowering of resistance to some other toxin. In optic neuritis the vision improves markedly, though not always completely, with treatment of the diabetes. In the palsies, all of the extraocular muscles have been affected, but palsy of one external rectus muscle is the most common lesion. The onset of these palsies is sudden and unlike the peripheral nerve involvements; it is painless. These usually clear completely with treatment of the diabetes.

In closing, I want just to mention the one ocular complication of diabetes with which we all became familiar as internes and which we used as an important diagnostic sign when dealing with unconscious patients seen on the Emergency Service: namely, the hypotony or softness of the eyeballs in diabetic coma.

Wainwright Building.

PULMONARY HEMORRHAGE IN TUBERCULOSIS AND THYLOQUINONE OR VITAMIN K.*

C. L. HARRELL, M.D.,
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and
A. C. RAY, M.D.,
Lynnhaven, Virginia.

I know of no symptoms more distressing than to see a patient hemorrhaging from the mouth. We usually have to sit by helpless praying that the bleeding will soon stop. As yet, no method has been devised, nor drug discovered, that offers immediate control.

Years ago it was considered good practice to administer a hypodermic of morphine; this seemed

*Read before the Norfolk County Medical Society, February 17, 1941.

to quiet the patient and check the cough, but in the end did more harm than good. It resulted in the blood settling in the lung, spreading the disease to other and healthy areas, often resulting in a tuberculous pneumonia.

Many preparations were put on the market, supposedly to hasten the clotting of blood; the author has tried them all from time to time, with disappointing results.

A few years ago when collapse therapy, in the form of pneumothorax, was first introduced, we thought we had an ideal treatment for pulmonary hemorrhage, but we soon learned that this form of therapy was applicable in only a small percentage of hemorrhage cases. First, it is not always easy to determine from which side the blood is coming. Secondly, if one could determine immediately which side was bleeding, in many instances the lung cannot be collapsed on account of pleural adhesions. Thirdly, one who is capable of collapsing the lung is not always available, so other and newer methods must be found.

The question naturally arises, what is the etiology of pulmonary hemorrhage It is due either to acute congestion or rupture of a blood vessel in the diseased area of the lung. The extent of the hemorrhage usually depends upon the size of the vessel ruptured. This does not answer the question entirely; why does one person with tuberculosis bleed, and another does not. In a small percentage of cases, hemoptysis is the first noticeable symptom, while another case may have tuberculosis in an advanced form for years and not even streak.

The percentage of cases that hemorrhage in tuberculosis is a debatable one. It is the authors' feeling that from 50 to 60 per cent of all cases expectorate blood sometime during the course of the disease. Dr. J. B. Nichols, of Catawba Sanatorium, feels that 20 to 25 per cent of patients hemorrhage as much as a dram or more and that 50 to 60 per cent will streak sometime during the course of the disease (personal correspondence). Dr. Frank B. Stafford, of Blue Ridge Sanatorium, feels that 50 to 60 per cent is about correct. At Tidewater Memorial Hospital there have been 291 admissions since the institution opened; 80 of these cases hemorrhaged while there or gave a history of having expectorated blood (36.3 per cent). This is much lower than I expected. There were 101 deaths, five of which died from a pulmonary hemorrhage, a fraction over 2 per cent, yet pulmonary hemorrhage is one of the most distressing symptoms of tuberculosis.

The question now arises, what is in the blood or make-up of the individual that does not bleed, which prevents him from bleeding, or what is lacking in the individual or blood of the patient that does hemorrhage. We will have to wait for the fertile mind of some biochemist to work this out. Phillip Jacobson recently suggested that many cases of spontaneous hemorrhage may be endocrine in origin; this remains to be proven[1].

Numerous articles have appeared in the literature in the past two years on the use of vitamin K in hemorrhage of biliary diseases and in hemorrhage of the new born, both prevention and control. I wondered if it might not be of some beneficial effect in pulmonary hemorrhage. I searched the literature but was unable to find a single article on the subject or where the drug had been tried. With this in mind, I wrote Squibb & Company for a supply of the new synthetic preparation, thyloquinone, for experimental cases in the Tidewater Memorial Hospital. They immediately sent me a large supply, free of charge. The result of this experiment will be reported to Squibb & Company. 2—Methyl—1, 4—Naphythoquinone, or Thyloquinone, is a synthetic preparation having the physiological properties of "Naturally Occurring Vitamin K" as previously reported in the studies of Dam and Schonheyder. They and others have demonstrated that vitamin K was a fat soluble constituent of green leafy plants, such as alfalfa, spinach and kale; it cures the anti-hemorrhagic diathesis caused by prothrombin deficiency.

Before using the drug, we decided to get the prothrombin time on a few cases of proven tuberculosis that had not hemorrhaged, to use somewhat as a control group; also, the prothrombin time on the hemorrhage cases before starting the drug. The laboratory work was done by the Misses Rawls and Stephens of the Doctors' Laboratory.

Technique: 2 cc. of venous blood was taken from the patient in a dry syringe and mixed with 1 cc. of sodium citrate, quietly agitated and taken to the laboratory. The test was always run within one to two hours after the blood was drawn, using the method of Howell.

Of the non-hemorrhage cases there were 16. Their prothrombin time varied from two to five minutes. Nine of these cases had a prothrombin time of four

minutes or below; only one of the non-hemorrhage cases had a prothrombin time of five minutes.

Of the hemorrhage cases there were thirteen. Their prothrombin time varied from four minutes to seven minutes. Nine of these had a prothrombin time of 4½ minutes or above, thus revealing by this method there was a slight deficiency of prothrombin in the blood of the hemorrhage cases.

You will observe that only 7 cases were actually treated with thyloquinone; all of these were given bile salts at the same time. It has been stated that vitamin K will not be absorbed except in the presence of bile. There was no gastro-intestinal upset and no toxic symptoms noticed. So far as we could observe no beneficial effect was noticed on the hemorrhage. Of course, this is too small a series from which to draw any conclusion. We will continue to give the drug a trial and hope others will do likewise. The control of pulmonary hemorrhage by the use of vitamin K, thus far, is not very promising.

REPORT OF CASES

Name: Mr. J. R. M.
Age: 33 years. Sex—male. Weight—92 pounds.
Date of onset: January 12, 1938.
Red count: 3,860,000. White count: 11,700.
Hemoglobin: 68%. Clotting time: 4 minutes.
1940:
July 3—Blood taken for determination of prothrombin time.
5—Prothrombin time—5½ minutes.
6—Hemorrhaged 3 oz.—2 oz.—1½ oz.—1 oz.
8—Began thyloquinone 1 mg. with 3 procholon tablets t.i.d. for three days.
12—No toxic symptoms incident to thyloquinone.
15—Second prothrombin time determine—4 minutes.
Red count—4,650,000. White count—14,700.
Hemoglobin—80%. Clotting time—3¾ minutes.
Nov. 13—Hemorrhage 2 oz.
Red count—4,340,000. White count—9,900.
Hemoglobin—70%. Clotting time—4 minutes.
Thyloquinone 1 mg. with 1 procholon tablet t.i.d.
14—Hemorrhaged 3 ounces.
Thyloquinone 1 mg. with 1 procholon tablet t.i.d. for three days.
17—No indications of toxic symptoms from thyloquinone.
24—Hemorrhaged 5 ounces.
Red count—4,820,000. White count—12,700.
Hemoglobin—70%. Clotting time—4½ minutes.
Thyloquinone 1 mg. with 1 procholon t.i.d. for three days.
No toxic symptoms.
No evident therapeutic effect of treatment.
25—Administered thyloquinone 1 mg. and 1 procholon tablet t.i.d. for three days. No toxic symptoms.

Name: Mr. J. M. S., Jr.
Age: 29 years. Sex—male. Weight: 129¼ pounds.
Date of onset: November, 1930.
Red count: 5,010,000. White count: 8,400.
Hemoglobin: 80%. Clotting time: 3¾ minutes.
1940:
July 15—Hemorrhaged 2 ounces.
Prothrombin time—3½ minutes.
Thyloquinone 1 mg. with 1 procholon tablet a day for three days (increased to t.i.d.).
18—No toxic symptoms. No further appearance of blood.
No appreciable effect.

Name: Mrs. E. G.
Age: 39 years. Sex—female. Weight: 169½ pounds.
Red count: 3,650,000. White count: 7,500.
Hemoglobin: 72%. Clotting time: 4¾ minutes:
1940:
July 1—Streaked.
2—Hemorrhaged 1 ounce.
3—Hemorrhaged 1 ounce—1 ounce.
4—Hemorrhaged ½ ounce.
5—Hemorrhaged 3 ounces—4 ounces.
6—Hemorrhage 1 dr.
8—Prothrombin time—5 minutes.
8—Started on vitamin K and bile salts. 1 mg. thyloquinone and 1 procholon t.i.d. for three days.
11—No toxic symptoms from thyloquinone.
15—Prothrombin time—4½ minutes.
Red count—4,300,000. White count—11,000.
Hemoglobin—80%. Clotting time—4¾ minutes.
22—Red count—4,410,000. White count—6,500.
Hemoglobin—80%. Clotting time—3 minutes.
Bleeding was abbreviated.

Name.: Mrs. B. H.
Age: 52 years. Sex—female. Weight: 86 pounds.
Date of onset: During 1931.
July 8, 1940:
Red count—3,750,000. White count—8,100.
Hemoglobin—72%. Clotting time—4 minutes.
1940:
July 7—Hemorrhaged 3 ounces.
8—Prothrombin time—4½ minutes.
Began thyloquinone 1 mg. with 1 procholon tablet t.i.d. for three days.
15—Second prothrombin time—5 minutes.
15—Red count—4,450,000. White count—8,800.
Hemoglobin—78%. Clotting time—3 minutes.
22—Third prothrombin time—4½ minutes.
Red count—4,750,000. White count—7,600.
Hemoglobin—85%. Clotting time—3 minutes.
Aug. 4—Hemorrhaged 3½ ounces—2 ounces.
5—Fourth prothrombin time—4½ minutes.
5—Red count—4,100,000. White count—8,800.
Hemoglobin—80%. Clotting time—3 minutes.

XUM

5—Administered thyloquinone 1 mg. with 1 procholon tablet t.i.d. for three days.
8—Fifth prothrombin time—4½ minutes.
Red count—4,220,000. White count—8,500.
Hemoglobin—83%. Clotting time—3 minutes.
Sept. 17—Hemorrhaged 2 ounces—1 ounce.
18—Hemorrhaged 1½ ounces.
19—Hemorrhaged 1 ounce.
19—Prothrombin time—4½ minutes.
19—Given thyloquinone 1 mg. with 1 procholon tablet a day for three days.
21—No untoward symptoms apparent from thyloquinone.

Name: Mr. V. J. M.
Age: 30 years. Sex—male. Weight: 165 pounds.
Date of onset: June 17, 1933.
Red count—5,200,000. White count—8,700.
Hemoglobin—90%. Clotting time—7½ minutes.
1940:
Nov. 2—Began streaking heavily; continued through 3rd and 4th.
4—Prothrombin time—6½ minutes.
Thyloquinone 1 mg. with procholon 1 tablet a day for three days.
5—Artificial pneumothorax began.
7—Heavy streaking checked after the third injection of air.

Name: J. T. (col.).
Age: 53 years. Sex—male. Weight: 110 pounds.
Date of onset: Spring of 1937.
Red count—4,580,000. White count—8,900.
Hemoglobin—80%. Clotting time—4 minutes.
1940:
Sept. 29—Hemorrhaged 3 ounces.
30—Prothrombin time—7 minutes.
30—Began thyloquinone 1 mg. with procholon 1 tablet t.i.d. for three days.
30—Hemorrhaged 2 ounces.
Oct. 1—Hemorrhaged 2 ounces.
2—Hemorrhaged 3 ounces.
Treatment had no appreciable effect on hemorrhaging.

Name: Mr. T. V. J.
Age: 35 years. Sex—male. Weight: 100 pounds.
Date of onset: February, 1934.
Red count—4,900,000. White count—10,300.
Hemoglobin—87%. Clotting time—2¾ minutes.
1940:
July 29—Hemorrhaged 2 ounces.
29—Prothrombin time—4½ minutes.
29—1 mg. thyloquinone with 1 procholon tablet a day for three days.
Aug. 4—Hemorrhaged ½ ounce.
5—Prothrombin time—5 minutes.
No toxic symptoms.
No appreciable effect.

Name: Mrs. K. B.
Age: 68 years. Sex—female. Weight: 175 pounds.
Date of onset: June, 1940.
Red count—5,600,000. White count—11,500.
Hemoglobin—90%. Clotting time—7 minutes.
1940:
Oct. 25—Began hemorrhaging—½ ounce.
26—Hemorrhaged—½ ounce.
27—Hemorrhaged—½ ounce.
28—Hemorrhaged—½ ounce.
28—Administered thyloquinone 1 mg. with 1 procholon tablet a day for three days.
29—Hemorrhaged—½ ounce.
30—Hemorrhaged—½ ounce.
31—Hemorrhaged—½ ounce.
Nov. 1—Hemorrhaged—½ ounce.
2—Hemorrhaged—½ ounce.
3—Hemorrhaged—½ ounce.
4—Hemorrhaged—½ ounce.
4—Administered 1 thyloquinone with 1 procholon t.i.d. for three days.
No results from thyloquinone.
Hemorrhage was checked by artificial pneumothorax.

In my opening remarks I mentioned we were helpless in hemorrhage cases. I do not want you to think we do not attempt to control the hemorrhage. Our method is as follows: As soon as a patient begins to expectorate blood, if he is not in bed, he is immediately put to bed in as comfortable a position as possible, preferably propped up on pillows at a 25 to 40 per cent angle disturbed as little as possible. We give some sedative, such as phenobarbital or bromides, endeavoring to quiet him. The patient is instructed to keep as quiet and as still as possible. An ice bag placed on the chest usually helps in this respect. Hemorrhage cases as a rule are highly nervous and very much disturbed. The nurse is instructed not to leave the patient any more than necessary and avoid crowding. One person should be in constant attendance while the patient is bleeding. Nature's process of controlling bleeding is the organization of a clot at the sight of the rupture; excessive coughing will disrupt this clot. Small doses of codeine or morphine should be given by the mouth to check excessive coughing, but not enough to abolish the cough reflex. I also give para-thyroid extract, 1 cc. hypodermically daily for three days and calcium by mouth. I do not believe this has any effect on large spontaneous hemorrhages, but has a very decided effect, at times, on those cases that bleed for several days. Occasionally one will find a bleeding case with an elevated blood pressure; this must be controlled either with nitroglycerine or large doses of barbiturates. I never use amyl nitrite, as I believe it does more harm than good. We keep the bowels open, preferably with salines, which helps

to deplete the vessels of fluids. Lastly, we resort to artificial pneumothorax where possible. In cases where the lung can be collapsed the hemorrhage usually is promptly controlled.

Often an ounce of prevention is worth pounds of cure. A large percentage of advanced tuberculous patients are confined to the house and chiefly in bed; they get practically no sunshine and eat an unbalanced diet. I wonder if feeding large quantities of citrus fruits, giving them an abundance of vitamin C, would help to prevent bleeding in a number of instances.

I am told that in our State sanatoria citrus fruits are withheld from the trays of hemorrhage cases for the reason they believe that a certain amount of citric acid will find its way in the blood stream as citrates and prolong the clotting time. Personally, I cannot subscribe to this therapy. Sailors used citrus fruits to stop bleeding from mucous membranes long before we knew anything about vitamins.

Mr. W. G. M., age 27, a bilateral case of tuberculosis, has been on the cure about three years, taking bilateral pneumothorax for about 2½ years. Both lungs are about 50 per cent collapsed; one would hardly expect him to hemorrhage. This young man is a bookkeeper. Long before he broke down with tuberculosis he worked in the basement, getting practically no sunshine. For the past year this man has been hemorrhaging about every four to six weeks—not a large hemorrhage but from heavy streaks to a dram or more, over a period of several days. It kept him and the family very much disturbed and caused me a good deal of anxiety. The first week in December, 1940, he began to expectorate blood in larger quantities than usual. He was given para-thyroid extract and calcium with no apparent result. He was then put on scorbutic acid, 50 mg. three times a day. His bleeding stopped in about 24 hours and he has not raised any blood since. At present he is taking 50 mg. twice a day and working.

In conclusion, I wish to state that, while we have tried vitamin K at the Tidewater Memorial Hospital in pulmonary hemorrhage cases, our results have not been encouraging. We expect to give it further trial and request that others do likewise and report their results. I also suggest that tuberculous patients be given an abundance of vitamin C in their diet in the form of citrus fruits and leafy vegetables to see if this will not lower the percentage of hemorrhage cases.

I wish to thank Miss Eleanor Rawls and Miss Betsy Stephens for doing the prothrombin times on these cases, Dr. A. C. Ray, the Resident Physician, and Miss N. Bethea Craft and her staff of nurses for administrating the drug and recording the results; and Squibb & Company for a liberal supply of the drug.

NOTE: Since reading this paper my attention was called to the article in the *Journal A. M. A.* by Dr. Henry C. Sweany and others of Chicago that supports the theory of a deficiency of vitamin C in tuberculous individuals.

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RÉSUMÉ OF DISCUSSION

I wish to re-emphasize one point of Dr. Harrell's paper. All equally diseased lungs do not bleed. To determine which lung will bleed and which will not is a very interesting and important piece of knowledge. The bleeding lung has a 25 per cent less chance of becoming a healed lung than the non-bleeding lung. Hence the importance of attempting to determine why some lungs bleed and some do not, so we may change the bleeding tendency if possible. At the moment we are attempting to determine the bleeding tendency by investigating the prothrombin time of the blood, and thereby the presence or absence of an adequate supply of vitamin K to the patient. Our greatest drawback has been that the result of the investigation in each case has not been a uniform rate of prothrombin time and a rate corresponding to the clinical condition. It shall be our effort in the future to determine the prothrombin time on each case as it is admitted, and from time to time to re-check the prothrombin time. With a comparison of the clinical status, we may determine the relation of the prothrombin time to the bleeding of the lung,

thereby determining the efficiency of any effort to eliminate the bleeders by means of synthetic vitamin K. We feel that if we may learn to convert the potential bleeder into a non-bleeder, we may have made a very substantial contribution to the treatment of advanced pulmonary tuberculosis.—A.C.R.

POST SANATORIUM TREATMENT OF PULMONARY TUBERCULOSIS.*

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Before discussing the care and treatment of tuberculous patients after discharge from the Sanatorium, it would seem appropriate first to review the various procedures which are followed in starting the treatment with these cases while in the Sanatorium. The two objectives to be desired in the two places are the same, but the measures applied in carrying them out must of necessity be different. In the Sanatorium the necessary facilities are available for most all of the special types of treatment, such as, collapse therapy to be started. Also, important features, such as, discipline and training in prophylaxis of tuberculosis—applicable in both places—are available. The Sanatorium with limited space for patients, and usually faced with a long waiting list, cannot hope to keep patients until they recover from a disease which is as chronic in its course as tuberculosis, but must discharge them after a few months stay to return home and there complete the treatment. Thus it will be seen the importance of considering jointly the treatment of the disease in the two places —home and sanatorium.

As tuberculosis workers we are challenged now more than ever before in our attempt to control this disease. We are startled by the fact that one out of every three young women who die between the ages of twenty and thirty years continue to die from tuberculosis. It is interesting to note in this respect that 44 per cent of the patients admitted to Blue Ridge Sanatorium, exclusive of children on the Preventorium, are between the ages of fifteen and thirty years. So we plead and plead more fervently than ever for more hospital beds for the tuberculous in locations where there are not enough of them provided to meet the need. In Virginia we are fortunate in having about two available beds for the treatment of white patients for every death, but in the colored the ratio is much less and is inadequate.

*Read as a part of a symposium on Tuberculosis before the Alexandria Medical Society, March 19, 1941.

In the campaign to eradicate the disease two prime objects must receive major consideration: first, the universal education of our people in regard to all phases of tuberculous disease; and secondly, the treatment of tuberculosis probably is the most essential factor which can be utilized in the control of this contagious disease. Until a short time ago many health authorities failed to realize that this was definitely a forward step in preventive medicine. The educational campaign, the case finding program, the early diagnostic campaign, the laboratory and X-ray, and many other activities have been of untold value. Nevertheless we cannot be very happy as to past results of this work since tuberculosis continues to kill more individuals in early adult life than any other disease, and, today in Virginia, stands second as a killer of our people, being surpassed only by pneumonia.

It is not only the death of those diseased which is embarrassing to us, but even more the ten other persons the individual may infect and who are in turn potential candidates to follow the same route.

Then we may say that at least a part of the solution of the problem would be to provide enough beds to hospitalize those so affected, and in addition to providing educational facilities in reference to the disease, and most important of all take immediate steps to render them non-infectious.

Regardless of whether tuberculosis is treated in a sanatorium or in the home, there are essentially three objectives to be accomplished if the best results are to be obtained. They are: (1) conversion of the sputum from positive to negative; (2) healing of the diseased process in the chest; (3) stopping tuberculous toxemia, thus enabling symptoms to subside, and as a result of this benefit the patient constitutionally.

It has long been known that a person with tubercle bacilli in the sputum is not only a public health

menace, but is a source of danger to himself. The infection may be spread by aspiration to other parts of the lungs, or it may extend to distant organs, thus causing complications. Tuberculosis of the larynx and of the intestine, particularly the former, is almost invariably secondary to pulmonary involvement, and is the result of a positive sputum. One reference will illustrate this: a short time ago we had occasion to go through our records on 1,200 cases discharged, and we found that of the patients admitted to the sanatorium with tuberculous disease in the larynx, 97 per cent had tubercle bacilli in the sputum. In contrast, of those admitted over the same period without involvement in the larynx, only 54 per cent had tubercle bacilli in the sputum. With these facts confronting us, it would seem that the logical procedure to follow in the management of these cases would be, if possible, to convert the sputum at the earliest possible moment. This not only would benefit the patient, but would safeguard against infecting those with whom he may live and come in contact.

In the earlier years of treating tuberculosis, the patients were put at bed rest with the hope that the pulmonary disease would heal spontaneously, and the sputum would become negative. In fact, not so much attention was given to the sputum, but the results of the treatment were based mostly on the gain in weight, improvement in symptoms, and clearing of the moisture in the lungs as revealed by physical examination. Today our methods are somewhat different. If there are any short cuts in the treatment, the patients want and should have them. The socio-economic status must have due consideration. The sooner these patients can be restored to health and an earning capacity, the better it is for the family, the State, and the communities in which they live. There must be a greater turn over of the patients in the sanatorium in order to provide beds for those needing treatment without having to wait unduly long to be admitted. Therefore, we have resorted to a routine which expedites treatment and offers the patient a better chance for recovery. This we call collapse therapy, or the surgical treatment of tuberculosis. Unfortunately not all cases are suitable for it, nor does it seem necessary or advisable to use it on certain types of cases, as they will usually make a good recovery without it. In no sense should it be inferred that the collapse of the diseased lung is all that is needed for recovery—other measures are very necessary and must be applied. *Surgery is a supplement to, and not a substitute for bed rest.* After the diseased part is properly collapsed, it does not mean that it is healed and ready for the patient to go back to work, or to do as he wishes. It means the diseased part, while still present, has been put at rest and placed in a better position to heal more quickly and more effectively if the other features of the treatment are properly carried out.

Various forms of surgery are used, and it is not always an easy matter to select the cases that are candidates for it, or to decide on the most suitable procedure for the individual case. Pneumothorax is the simplest and the most effectual procedure of any now in use, and it is our practice to try this first. If pleural adhesions are too dense and numerous to permit proper collapse, then we give serious consideration to the advisability of attempting something else, such as extrapleural pneumothorax, phrenic nerve operation, thoracoplasty, etc. Approximately 70 per cent of the patients admitted to this Sanatorium with definite tuberculosis are selected and tried on pneumothorax. With the aid of intrapleural pneumonolysis we succeed in satisfactorily collapsing about 55 per cent of these cases. "Open" cases, that is, those with tubercle bacilli in the sputum are mostly selected, provided they are otherwise suitable. The exception to this rule would be those with slight pulmonary disease and negative sputum, but, after a short period of observation on bed rest, the lesion on X-ray has not shown satisfactory improvement, or the symptoms have increased.

This departure from our former routine has given us gratifying results. Last fiscal year only 6.6 per cent of our patients at the time of discharge had tubercle bacilli in their sputa as compared to 34 per cent on discharge ten years ago. The percentage is about as low as can be hoped for because there will always be a group of obstreperous and homesick patients with positive sputa who will not stay in an institution long enough for conversion of the sputum, and who will be a public health menace wherever they may be. Also, there is a group of hopelessly advanced cases with positive sputum for which nothing can be done, and obviously they cannot be kept in the sanatorium indefinitely when more suitable cases are waiting to be admitted.

In reference to home treatment, it may be said that most of the measures started in the sanatorium

should be continued after discharge. In addition to admission and intermediate reports, our policy at the Sanatorium is to give the physician who sent the patient to us a full discharge report, and to advise the patient to return to that physician. This report includes chest findings on discharge, results of treatment, and suggestions for after care and treatment of the case. Through this procedure conflicts on home treatment are avoided between family physician and well-meaning friends and relatives of the patients. All cases taking treatment at home should be under the care of a physician, and routine visits to the patient's home should be made by him. This is just as important as it would be for the physician to see regularly a chronic cardiac case. It is true tuberculous patients need very few drugs, but the psychological effect on the case of seeing the physician regularly is very stimulating and quite beneficial. Physical examinations and medical supervision are also essential to the health and safety of the patient and should be continued for years after the strict treatment for the disease has ceased.

If pneumothorax has been started while in the sanatorium, there will be available in most every part of the State a qualified physician who can administer refills so that patients may continue with this collapse therapy. At the present time, according to the report of the Director of the Tuberculosis Out-Patient Service of the State Department of Health, there are fifty-six such stations over the State, and 510 patients are receiving treatment from this source.

Some system should be adopted for routine sputum examinations. Perhaps for the average case under home treatment, once every two months would be sufficient. It should be remembered, however, that the sputum findings are not entirely reliable in known cases of tuberculosis, because one can never tell just when some condition may arise which will cause it to become positive again when it had on several occasions previously been reported as being negative. In this way it may give a patient a false sense of security, and perhaps cause a relaxation of precautions and probably endanger the lives of the other members of the family. The patient should understand that the safest procedure to follow is to be careful at all times, regardless of the report from the sputum examinations.

A routine schedule of bed rest, including both physical and mental relaxation is very necessary, but is often quite difficult to enforce due to the various disturbing factors which may develop in the home. This can be minimized if the family understands and cooperates with the physician and nurse in carrying out the prescribed schedule.

Written instructions as to the time to be spent in bed and when visiting is to be permitted will assist those charged with maintaining discipline. An afternoon "quiet period" of at least two hours, preferably between two and four, should be observed. During this time there should be no visiting with the patient, and the house should be gotten as quiet as possible and anything else avoided which is apt to be disturbing. Meals should be served regularly, but like many other details it need not interfere with the routine in the home. If there are children, the ever-present danger of infecting them exists. A hard and fast rule to make is that children must not come into the patient's room, or in contact in any other way with the patient.

Diet, while not as important as rest, must be given due consideration in outlining the course of treatment. It is no longer necessary, or considered advisable to force feed or "stuff" the tuberculous patient. The diet should be appetizing, well balanced, high in vitamin content, and should contain sufficient calories for the patient to show a steady gain in weight. Obesity, however, is no longer a prerequisite for recovery. Our aim is to get the patient up to standard weight, and then regulate the caloric intake so that the weight will be maintained without further gain. Our records show that 80 per cent of the patients coming to the Sanatorium have "indigestion", or digestive disturbances in one form or another. Therefore, it is necessary to have in addition to the regular menu, a list of special diets to be used for those cases requiring it.

The question of when walking exercise should be started is an individual problem, and should be decided primarily on the merits of the case. A good general rule to follow is to permit no exercise until all symptoms which denote activity have been absent for at least two months. When exercise is permitted, it should be a very gradual process indeed, and increased only after being convinced that it is doing no harm and is being well tolerated by the patient.

There are numerous other important features in home treatment of tuberculosis which might be discussed here, but time will not permit. It may be

said in conclusion that regardless of the time and effort put forth on the part of the doctor and nurse, and the schedule prescribed, the results are not going to be satisfactory in certain types of cases due to lack of cooperation on the part of the patients. The successful treatment depends in a large measure on the intelligence of the patient and his or her willingness to obey instructions. Someone has aptly said: "The successful treatment of tuberculosis is three-fourths above the collar bone."

CHANGES CAUSED BY CEREBELLAR NEOPLASMS IN THE ELECTROENCEPHALOGRAM

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During recent years an advance in Neurology and Psychiatry has been the application of a vacuum tube, condenser coupled amplifying system known as the electroencephalograph, to the detection of electrical activity from within the skull. This instrument picks up voltage in the order of millionths, amplifies it and records either on bromide paper or ordinary folded paper, depending on whether mirror or ink-writing oscillographs are used.

In this early, but intensive, stage of investigation with the instrument certain characteristics have been found which delineate normal neuronal activity from abnormal. With the patient at rest and eyes closed, the usual, normal tracing may be described as follows: from the occipital region sinusoidal waves appear at a frequency of 8 to 14 a second and 50 to 60/1,000,000ths volt (yv) maximum (alpha waves). Opening the eyes will cause the waves to disappear. They tend to arise in clusters, having a crescendo-diminuendo effect. As the electrodes are moved anteriorly, these waves tend to disappear, being supplanted by faster frequencies, 15 to 40 a second and 15 to 30/1,000,000ths volt maximum (beta waves). These waves are prominent in the parietal and frontal regions. See figure 1.

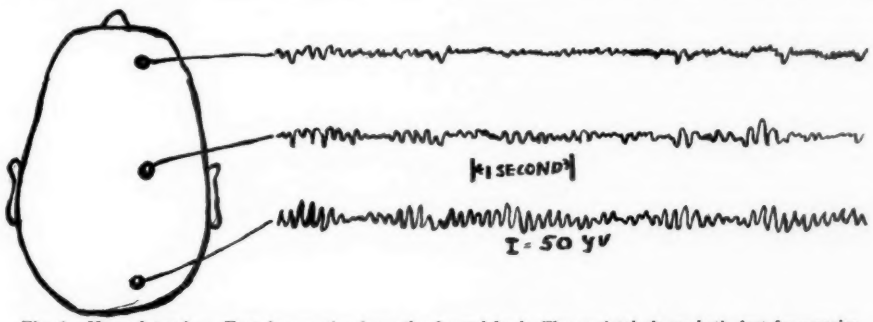

Fig. 1.—Normal tracing. Fast frequencies from the frontal lead. The parietal shows both fast frequencies and waves of the alpha type. The occipital lead shows alpha waves and the "Berger" rhythm.

The above description is somewhat classic because each individual has his peculiar pattern. Nevertheless, those waves are to be contrasted with abnormal neuronal activity. There have been evolved three criteria for determining abnormal "brain waves". These are (1) frequencies being slower or faster than normal; (2) voltages being greater than normal; (3) a combination of these two. Epilepsy, a disease which has been widely studied, produces during grand mal seizures, waves which are faster and of greater voltage than normal while during petit mal attacks slower than normal and faster than normal wave alternate. Both types of waves are of very high voltages. Tumors, scar tissues, and cysts produce no measurable bioelectric activity themselves, but because of disturbing the functions of the cells about them, abnormally slow, high potential waves arise on their margins. This feature of neoplastic growths in cortical areas is used to demonstrate their size as well as their location.

These descriptions have been worked out for

lesions and disturbances of the cortex. This is to be expected as the electrodes are placed on the scalp, not in areas within the skull. Experimental and clinical investigations of deep structures are relatively few from an electroencephalographic standpoint. Smith, Walter and Laidlaw in 1940 reported tracings taken from eight extracortical tumors, six of these being cerebellar. They found changes in the occipital region and suggested they were "the result of secondary damage to the occipital lobes produced by pressure exerted by the lesions upward through the tentorium". Their electrodes were applied to the scalp, not to deeper tissues as might be inferred.

The following report is based on two cerebellar neoplasms which were suspected from changes found in the occipital poles by the electroencephalograph. They were studied after operation by this instrument.

CASE 1. Colored male, age 11, was admitted to the University of Virginia Hospital with the complaint of headache for three years. For several months prior to admission he staggered in walking and had occasional attacks of vomiting. His psychomotor activity had become greatly retarded.

Examination revealed a well developed and nourished colored boy; circumference of head 58 cm.; exophthalmus in both eyes; pupils dilated; right disc blurred, left had distinct margins; retinal veins were engorged and tortuous; no nystagmus; other cranial nerves normal; all reflexes were equal and active; no pathological reflexes; gait ataxic; romberg positive; slight adiodokokinesis; ataxia in finger to nose test; visual fields restricted.

Wassermann and Kahn negative; ventricular fluid, hemocytology and urinalysis normal. Skull plates showed separated sutures and blunting of the clinoid processes. Ventriculogram demonstrated a very large internal hydrocephalus.

At operation by Dr. J. M. Meredith, an atypical medulloblastoma which arose in the mid-line and extended into the left cerebellar hemisphere was found. Following as complete removal as possible, he was given 5,850 R units of X-ray through three channels to the posterior portion of his head.

CASE 2. Colored female, age 9, was noticed to have a staggering gait for a year prior to hospital admission. She developed occipital headaches, and as these symptoms progressed she would vomit with the headaches.

Examination revealed a well developed and nourished colored girl; the head was considered large; cranial nerves were normal except the optic which showed 1 to 2 diopters of choking; no pathological reflexes; hyporeflexia; suboccipital tenderness; staggering gait; cerebellar tests of the extremities done poorly; romberg positive; nystagmus to right and left.

Wassermann and Kahn negative; urinalysis normal; ventricular protein 10 mgm.; X-ray showed the suture lines to be wider than normal and the digital markings to be increased.

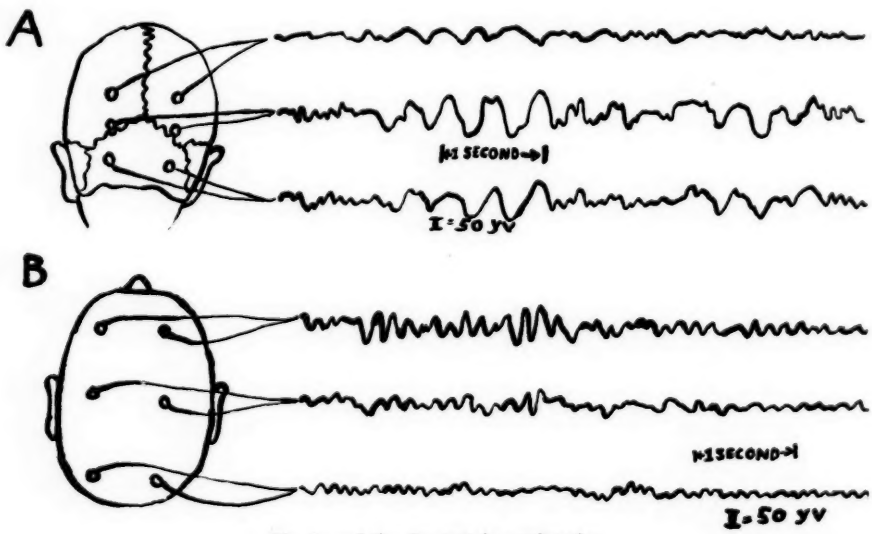

Fig. 2a and 2b.—See text for explanation.

At operation Dr. Meredith found a cyst the size of a duck's egg in the right cerebellar hemisphere. In the lumen of the cyst, which protruded into the fourth ventricle, there was a hemangioblastoma. To date the child has received 3,000 R units of X-ray to the posterior portion of her skull.

Electroencephalograms in Case 1, taken prior to operation, show the greatest changes (highest poten-

tial and slowest waves) 5 to 6 cm. above the inion. These recordings were made with the electrodes in a frontal plane and were the composite bioelectric activity of both sides (bipolar recordings). As the electrodes were moved downward or upward from this area, the potentials became less, indicating they were being moved away from the point of maximum voltage. Figure 2a.

Twenty-four days following operation another tracting was made. Figure 2b. This showed almost normal alpha activity in the occipital region but in the frontal region there arose groups of large slow waves which had not been present. This indicates damage in the area, possibly the residium of the internal hydrocephalus.

A tracing made fifty-eight days after operation showed irregular 3 to 7 a second waves of 100 yv maximum amplitude in the occipital region. From the parietal region there arose fairly regular 5 to 7 a second of 50 to 100 yv maximum amplitude. The frontal region showed about the same fast frequencies and a few 5 to 7 a second waves. These large slow waves originated, on careful examination, from each parietal region, 17 cm. above the inion. The change represented in this post-operative tracing, with faster synchronization and lower voltage is toward more normal activity but indicates damage to the cortical cells. Figure 3b.

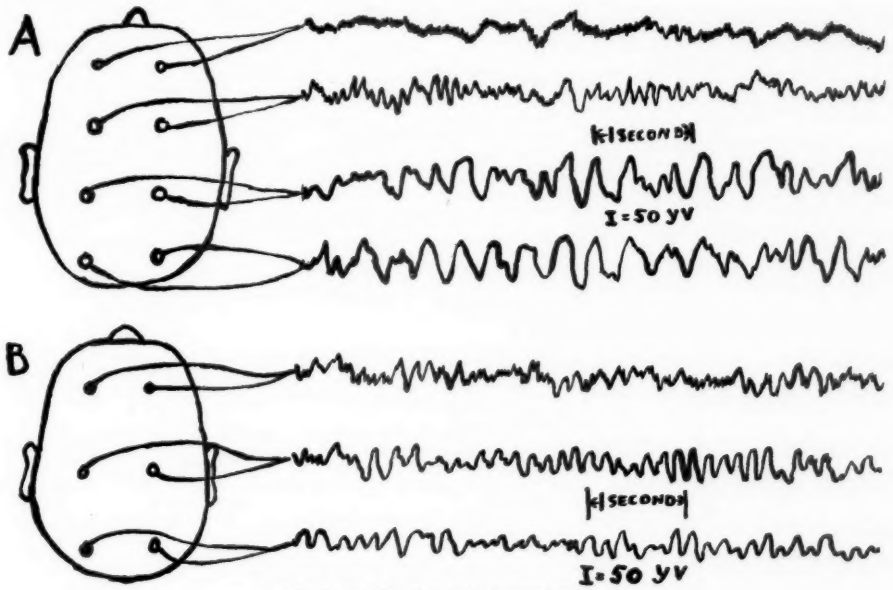

Fig. 3a and 3b.—See text for explanation.

Case 2 was studied more thoroughly. Measuring over the vertex from the inion to the bridge of the nose, the distance was 42 cm. Leads were placed in the frontal plane as in the first case and the greatest abnormalities were seen 7 cm. above the inion. At 14 cm. above the inion there was slight diminution in voltage; at 22 cm. the frequencies were faster and lower in potential and at 32 cm. fast frequencies were pronounced. Figure 3a. In order to locate as precisely as possible the area of maximum potential, triangulation was resorted to, which placed it 7 cm. above and 2 cm. to the left of the mid line, from the inion.

COMMENT

When the spherical spread of such electrical waves are taken into consideration it can not be stated from the tracings whether the source of this abnormal bioelectric activity is in the cerebellum or occipital lobes. In Figure 3a it may be seen that, although there is a difference of 7 cm. between the occipital and the next higher pair of electrodes, there is definite, yet little difference between the curves. This is due, in all probability, to two factors. One is the spherical shape of the skull. The other is dependent on this. If the origin of the abnormal activity is deep seated the radii to both pair of electrodes are about the same. Finding the point of maximum potentials in-

dicates the shortest radius to the source of abnormality.

One might suppose from these considerations that geometric analysis or scaling of the potentials as they diminish with distance from the origin of the activity would give this position away. The problem and proof is not so simple.

In the cases of Smith, Walter and Laidlaw there was no change in the slow waves that could be attributed to opening the eyes, which ordinarily abolishes alpha activity. There was slight attenuation of these slow waves but they promptly returned to their former configuration. The alpha activity which was present was affected to a greater degree. Although their brain stem tumors gave bioelectric changes in occipital leads, there is a report by W. G. Walter of a diencephalic and hypothalamic tumor which did not do this. The changes reported were those of sleep and it is possible that such potentials could obscure any localized changes.

That the changes are not due to increased intracranial pressure alone is shown by the fact that other causes for increased pressure do not cause occipital abnormalities. It would seem that the intracranial hypertension has a damaging effect on the cortex. In both post-operative tracings there are areas away from the occiput which show abnormal potentials and frequencies. In the cases reported here they have been present two months, in those reported by Smith, Walter and Laidlaw six months.

The question would seem to be open as to whether the changes noted on the electroencephalogram are due to changes in the occipital lobes or cerebellum. When abnormalities as described, occur in the occiput they certainly raise the question of cerebellar neoplasm.

SUMMARY

Two cases of posterior fossa neoplasm with their pre- and post-operative tracings are presented. These tracings are suggestive but not diagnostic of cerebellar neoplasms.

THE PROBLEM OF THE PSYCHOPATHIC PERSONALITY IN THE FEEBLE-MINDED INSTITUTION.*

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The title of this paper is "The Problem of the Psychopathic Personality in the Feeble-minded Institution", but, for the purpose of my discussion, I am going to confine myself specifically to the very real problems presented by that type of psychopathic personality that *we* call the "constitutional psychopath".

Like any other institution caring for the mentally ill, we have in our patient population at least one or two examples of every known type of psychopathic personality, ranging from the intensely irritating paranoid psychopathic personality of the so-called typical epileptic deterioration, to the pathetic psychopathic inferiority. But no one of these psychopathic personalities presents as many or as grave problems as the "constitutional psychopath".

I must confess that it is with some fear and trembling that I present this term to describe a specific type of individual. In the past it has been used more or less as a "shotgun" diagnosis to describe a person who was not normal, but who none the less could not be classified under any of the standard diagnoses. Nevertheless, we feel that the term has a definite place in the nomenclature; that far from describing any and all vague mental complaints, it should be used (as we used it at the Lynchburg State Colony) to define a specific type of mental disorder. And it is in an attempt to make clear the syndrome presented by our "constitutional psychopath" that this paper is written.

About one hundred years or so ago—or maybe much further back—the term "moral imbecile" was coined to describe certain people who were not insane, epileptic, or feeble-minded, but who were nonconformists to the extent that they could not possibly be considered normal mentally.

Somewhat later the "moral imbecile" was replaced by the "constitutional psychopath"—and by many other ponderous, descriptive terms.

*Read before the Neuropsychiatric Society of Virginia at its winter meeting in Richmond, January 29, 1941.

But nowhere can one find two definitions of the term "constitutional psychopath" that are identical, and it would seem that almost no two psychiatrists view the term in exactly the same light.

For instance, a couple of weeks ago, I attended a seminar that was held in Washington for the psychiatrists who were to advise the local Selective Service Boards. Inevitably the question of the advisability of inducting into the Army those men who might variously be described as "moral imbeciles", "psychopathic criminals", "recidivists", etc., was raised. Dr. Paul Schroeder stated that a recent survey of the penitentiaries in the United States revealed the fact that the authorities of one institution classified 90 per cent of their criminals as constitutional psychopaths, while the authorities of another penitentiary stated that only 3 per cent of their prisoners were constitutional psychopaths. The other penitentiaries gave percentages ranging somewhere between those two widely divergent figures. From this it is easy to see that the interpretation of the syndrome represented by the term "constitutional psychopath" has been anything but rigid. Certainly the criminal population of the penitentiaries of two different states cannot actually be that different.

At this same seminar Dr. Simons asked Dr. Hall —who was conducting the meeting in question—to tell the psychiatrists assembled there what they, in turn, must tell their local boards about the constitutional psychopath. He asked that, in a nutshell, they be told what to look for so that they might easily recognize the constitutional psychopath. Dr. Hall gave his definition of the term; then called upon four or five more nationally known psychiatrists. Each one's definition varied in certain vital points. Finally, Dr. Sullivan was asked to express himself. He made little attempt to outline the syndrome, but told the psychiatrists that in the long run the diagnosis must be made from the history of the patient.

At the Lynchburg State Colony we call a patient a constitutional psychopath only if:

1. His intelligence quotient as measured by a standard psychological scale or battery is at least 70 per cent; his mental age at least ten years; in short, he cannot be regarded as feeble-minded *per se*. At this point I should like to mention the fact that the Army has recently gone on record as stating that any man whose mental age is at least ten years (and who is otherwise sound) will be acceptable for military training.

2. He has demonstrated by his conduct in the past that he respects neither the personal nor the property rights of others.

3. He has shown—again by his past conduct—a subconscious immutable fixity of purpose to compel society to conform to his wishes. It is noteworthy that there is no systematic chain of delusions.

4. He has shown—again his history is the informant—that while he can exercise fair judgment and reasoning on the immediate, he can never view a problem on a long-range scale. That is, for example, while he may be uncommonly clever in the execution of a theft, he will never see what the ultimate result of his act might be either to himself or to others. He simply cannot see that he must bear his share of social responsibility.

5. He has exhibited an intense hypersubjectivity —a selfishness that excludes any consideration, even for those who should be dearest to him by reason of kinship or friendship.

6. He is not classifiable as epileptic or insane, and his syndrome is not the result of mental disease or trauma.

I am now going to read you thumbnail sketches taken from the records of six of our constitutional psychopaths—these have been picked at random from the fifty or sixty of these individuals that have been sent us in the past several years.

CASE NO. 1: B. S. Admitted to the Colony July 17, 1930. B's Father was a borderline case of mental deficiency; his Mother was intensely neurotic. B was the only child—all his life he had been badly pampered and spoiled. So far as we could ascertain, he had received no real discipline. His Father had spanked him once, but had apparently felt thereafter that he should never again raise his hand against his child. In short, B's heredity and environment were both bad. When he came to the Colony he was only eight; but he had already committed many misdemeanors: he had sold his parents' furniture to a junk dealer (while they were away from home); he had even managed to abstract a couple of dollars from the pocketbook of the secretary of the psychiatrist who was examining him—without either the doctor or the secretary being aware of the theft. He had been expelled from school as utterly incorrigible. Although the diagnosis of constitutional psychopathic inferior personality had been

made at one of the State's leading private sanatoriums, B was sent to the Colony as feeble-minded. Mental examination revealed that the boy had an intelligence quotient of at least 95 per cent—his personality, however, fitted our interpretation of "constitutional psychopath" to a "T". He was glib, quick, markedly egotistical, and just about as fresh a young'un as one could hope to see. He stayed with us for five years. During that time he never caused any serious trouble—because he was too closely watched! But he was forever creating disturbances among the other patients, and he would leave the Colony with the speed of a frightened jack rabbit if given the slightest opportunity. He certainly did not belong at the Colony and when Mr. S. requested permission to remove him, expressing certainty as to his ability to control B, we discharged B. Since then, I have been told, he has been in the Courts on many occasions. He has been a patient at the Eastern State Hospital, and he is now in the Criminal Insane Department of the Southwestern State Hospital!

CASE NO. 2: F. S. (no kin, by the way, to B. S.) was admitted to the Lynchburg State Colony on the 15th of March, 1939. F came, so far as we could learn, from a middle class home of the average urban type. There was no record of mental disease or defect among his relatives. He had attended school, very irregularly (as he told us, he was more interested in play than in education) for some ten years and had never progressed further than the third grade. When he was sent to us at the age of 18 he had already been in jail eleven times, in the Industrial School three times; once at one of the State hospitals for a period of observation. The committing authorities sent F to us as feeble-minded—they could not believe that anyone could possibly give so much trouble and not be feeble-minded! On mental examination at the Colony, however, we found that he had a mental age of eleven years. While he was with us—and as long as he was under the closest sort of personal supervision from his attendant—he gave no real trouble: once the attendant's vigilance was relaxed, however, F would be off like a shot. He played third-base on our softball team and did a splendid job of it. He was a likable, friendly sort of chap—so long as that attitude best served his purpose! Finally, he made good his escape from the Colony; shortly afterward he was arrested in North Carolina for stealing automobiles; he was convicted in the Federal Court and is now serving a three-year term at Chillicothe.

CASE NO. 3: F. H. Admitted to the Colony on the 13th of February, 1937. F's Father was alcoholic and syphilitic; his mother was mentally deficient; we have one of F's younger brothers (feeble-minded) at the Colony—six other siblings of F's are said to be subnormal mentally. However, in early childhood F was taken from his own home and was placed in a good foster home in one of the rural counties. He was raised by thoroughly good foster-parents. F gave his foster-parents much worry by his refusal to apply himself at school. As he grew older he became gradually more unmanageable; and when he finally became aggressively interested in small children of the opposite sex his foster-parents had him committed to the Colony. On examination we found that he was of about dull average intelligence. We felt that something might be done with him, and we put him to work in our central office as an office-boy. For a short time he did quite well; then one evening he stole the keys to the psychologist's car. That night he, and another boy whom he inveigled into accompanying him, went to Lynchburg to Mr. Buck's home, confidently expecting to be able to abscond with his car. But to his chagrin, F found that the car he had hoped to steal was locked in the garage and he couldn't get it. F had the good fortune (for him) to find a car across the street parked with the motor running! F drove that car to Amherst where it broke down. Under pretence of wanting to buy a second-hand car, he persuaded an automobile dealer in Amherst to permit him to try out a car—he drove that auto to Alexandria, where it, too, failed him. He then stole a third car: he was arrested in Maryland while driving that. F was tried, convicted, and sent to the State Farm. After he had completed his 18-months sentence there, he was released. He promptly stole several more cars, and led the police a merry chase from one end of Virginia to the other before they finally caught him. At one time he actually went so far as to return to the Colony and steal a set of license plates from the car of one of our staff physicians. F is now serving a long term in the penitentiary.

CASE NO. 4: G. P. Admitted to the Colony on the 20th of July, 1940. G's Mother was a woman of better than average intelligence and background.

She had graduated with honors from an outstanding woman's college after majoring successfully in music. G's Father—a most eccentric man—was a successful tailor. G's Mother divorced his Father, when G was but a small boy, on the grounds of cruelty. G's Mother died when G was but nine, and the rest of his early adolescence was spent in one foster-home after another. He reached the fourth grade in school. He was in constant difficulty with the school authorities, and others, from his early teens onward. When we got him, he was 26 years of age. He had been in a mental hospital under observation. He had been at the Industrial School; and had been jailed several times. He came to us from the State Farm. They sent him to us, not because they thought him feeble-minded, but because they felt he should not be at large and they didn't know where else to send him. He had lived all up and down the Eastern Seaboard. He had never had any trouble getting a job, for he made a rather good impression; and he had never had any trouble holding a job until he either stole something or his homosexual tendencies got the better of him—as they frequently did—and he made advances. On mental examination, we found out that he could not possibly be considered feeble-minded; on the contrary, he had about average intelligence. But his personality was very badly warped. He said with sullen pride, "They never *caught* me in the act of committing sodomy." He frankly confessed to a taste for fellatio and spoke of it as a "habit". After G had been here a short while we told him that he didn't belong here; that we would discharge him. He begged so piteously to be allowed to spend the winter here, that we agreed to permit him to stay. We put him to work in our dairy barn. Our herdsman reported that he could not ask for a better hand. G, however, as we found out afterward, was harboring a bitter grudge against the State because of a whipping he had received at the State Farm. He reasoned that if he destroyed something at the Colony, he would thereby secure revenge against the State. So he repaid our kindnesses to him many fold by setting fire to our larger dairy barn and doing damage to the tune of ten thousand dollars! We turned G over to the Amherst County authorities. He was tried in Amherst Circuit Court, convicted of arson, and sentenced to ten years. And—poetic justice—he is now back at the State Farm that he so despised.

CASE NO. 5: B. B. Admitted to the Colony on the 10th of April, 1937. B's family history is essentially negative. Her father owns one of the better farms in one of the Southside Counties. Her raising was good. B reached the sixth grade in school after six years' attendance. At the onset of puberty, she began to give trouble. Her family soon found it quite impossible to keep up with her—not unnaturally they felt she *must* be feeble-minded (else she could hardly be so thoroughly uninhibited and friendly), and they sent her to us when she was 15. Fortunately she had escaped pregnancy. Mental examination revealed a mental age of above 12 years; she had many grave personality flaws. After she had been sterilized we permitted her (at her family's request) to leave on parole. She was returned in the next few days, however. She has now been with us almost continuously for the past 45 months. During that period she has had to be disciplined for various infractions of the rules no less than 43 times!

CASE NO. 6: M. H. Admitted September 20, 1940. M's Father left her Mother when M was very small. M was raised by her grandparents. They did the best they could for her, but they were never able to control her well. M reached the third year in high school; she was always a rather good student. She was in difficulty, though, from her early 'teens onward. She was finally expelled from high school because of her sexual promiscuity. Soon thereafter she married a man who was feeble-minded. For a while this seemed to mark a turning point in M's life. She seemed contented to support her husband, and she got into no more trouble until she became pregnant and had to leave work. She then promptly abandoned the new leaf to which she had turned. She appealed to the welfare agencies and was given money and supplies by them; but when they found she was using the money to pay her way into the movies and selling the supplies they furnished her, they soon withdrew their aid. She then took to begging, even from negro churches, and finally she took to forgery. When she was sent to us by the Court, she stood indicted on seven distinct charges of forgery. We found that she had a mental age of better than 13½ years. She made an excellent first impression; she was quick, alert, and quite willing to admit her guilt—once she was certain the examiner knew all about her. She stated (in clas-

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sical fashion) that she had learned her lesson; that never again would she sin; but at no time did she exhibit any real penitence or any real understanding of the possible punishment that faced her. She was in trouble of one sort or another the entire time she was here. We returned M to the Court for trial. The Court saw fit to release her with a suspended sentence. I am sure that we have not heard the last of her!

These six patients are good examples of what we call a "constitutional psychopath". Though their cases differ in certain respects, each qualified as a constitutional psychopath under the six essential points I outlined before.

If one were to take a composite photograph of all the male constitutional psychopaths and another of all the female constitutional psychopaths that we have ever had, he would have photographs of a couple who might easily pass as Mr. and Mrs. Average American. In other words, as a rule, there is absolutely nothing in the external appearance of the constitutional psychopath that gives a hint to what is going on underneath.

But, you will ask, what makes the constitutional psychopath what he is. It is dangerous to generalize, but if one were to compile statistics on the history of all constitutional psychopaths he would find that the majority of them (there are exceptions to this rule, of course) had poor heredity, or poor environment, or both. By "poor environment" I do not mean *material* poverty as much as *cultural* poverty. Only too often, I believe, these people have no respect for the rights of others because their parents before them had no respect for law and order. Then, too, if, when they were children, they could not respect their own parents, whom, as adults, might they respect. Only too frequently, their fathers have been drunkards and thieves, their mothers prostitutes and sots; their homes have been homes in name alone.

Some of the constitutional psychopaths, having no real standards to follow and often seeing their own parents flout the customs of the land finally grow to believe that only fools will conform. Others, though the parental example is of the best, soon become exasperated at their failure to succeed by normal methods and conclude that the modes and customs of society are silly. And, in any event, being definitely weak personalities to begin with, they soon become sorely frustrated individuals. The restrictions of society become unseen fetters to them—fetters, however, that must be broken lest their egos suffer a defeat that they cannot admit; there is nothing for them to do, as they see it, but wage active combat with the rest of the world. Unable to admit the possibility of defeat, they cannot sense the inevitability of ultimate apprehension and punishment when they embark upon a course of strict non-conformity.

When caught they will cheerfully admit their guilt —if, as they usually do—they shrewdly sense that by doing so they are the more likely to be dealt with leniently. Like the chronic alcoholic, they will beg for "one more last chance"—but unlike the alcoholic, they apparently can never feel, even momentarily, anything akin to real penitence: their only desire is a chance to try it again, vowing that this time they will not be caught. Never for a moment do they feel that anything that happens may have been the result of anything but blind luck upon the part of the enemy, society.

In their persistence at non-conformity they resemble the post-encephalitic behavior problem patients: but the crime of the true constitutional psychopath is not as impish, senseless or impulsive as that of the post-encephalitic. The constitutional psychopath will usually exercise considerable skill in his efforts; they will be the result of reasoning, and the misdeed will usually be more in line with what one would expect of one of this intelligence level.

He is often committed as feeble-minded by the physician inexperienced in mental cases because the physician cannot understand how anyone could do what the patient has done and not be feeble-minded: and then, to top it off, so to speak, be so supremely self-confident. Actually, of course, that "self-confidence" is nothing but a protective armor that the patient has donned. He'll never let the world know that it has him down.

But the fact remains that patients of this type *are* sent to us and far too often. They do not belong to us; they are not feeble-minded; and there is not a thing that we can do to help them. They are a continual source of trouble—they do not declare an armistice in their fight with society simply because we have them in custody. On the contrary they take a particular delight in attempting to proselyte our lower-grade, essentially benign patients, to whom they are glamorous individuals. The constitutional psychopath is forever trying to get away from the

Colony himself—but, not content with that, he will almost always take some other patient (or patients) with him.

We cheerfully concede that the constitutional psychopath belongs in an institution—but why pick on the Lynchburg State Colony?

At the moment, there is no institution in Virginia, either mental or penal, to which he really does belong. I believe that the State could save itself no end of time, money, and trouble, if it would build an institution of the farm colony type to which these constitutional psychopaths could be sent for an indeterminate period. Such a farm colony could be nearly self-supporting, and it could certainly be made a fairly attractive place. If we are to keep these people permanently out of society, it is only fair that we permit them to lead as nearly normal lives as possible while they are in custody. Some will say, "Oh, yes, that's all very well, but what if someone is simply railroaded into such a Colony." That such a thing might happen is, of course, quite possible—but anyone *not* a constitutional psychopath should not have much difficulty convincing the Colony's resident psychiatrist of that fact.

We are agreed, I think, that these people must be kept out of society. But they must also be kept out of the hospitals for the insane and the hospitals for the feeble-minded and epileptic where they do untold harm. The sooner a farm colony is erected for the constitutional psychopaths and for them exclusively, the sounder many of us will sleep.

DISCUSSION

DR. D. C. WILSON, Charlottesville: This paper of Dr. Arnold's is most comprehensive and delightful in its arrangement, as well as in presentation. This paper is a sequel to that of Dr. Blalock given before this Society at Virginia Beach. Dr. Arnold's definition cannot be improved, but the problem before us is not so much one of definition, but of finding a dynamic approach to the problem that will lead to some sort of practical solution.

Eugene Kahn, writing approximately twelve years ago, distinguished the psychopath by means of a quantitative lack at each level of integration. Certainly the psychopath has a lack of development which makes his inability to get along, but the knowledge of this lack has not helped us so far in our effort to solve the problem. Karpman approaches the problem from the analytical standpoint, and claims to have cured such patients by this method. He urges that the category "Psychopathic Personality" be eliminated, as these individuals are not essentially of one class, but fit into many other types of disorder. There is also a tendency among analysts to shift the burden of the fixation in this type of disorder from the mouth to the anus; certainly a major change in approach, but not one that will help us much in the practical handling of the numerous psychopaths.

Curran's work reported before the State Mental Hygiene Society last fall offers more than anything produced in some time. The report of his follow-up is out, and also an analysis of his work by Schilder has been published. He claims 67 per cent well after four years, and this after only four weeks stay in the ward at Bellevue. By means of their individual psychotherapy they seem to give insight to their patients and then by group psychotherapy they induce a respect for authority, as well as a desire for a new mode of living, a marvelous accomplishment that undoubtedly should be the aim of all similar forms of treatment. If we are to treat psychopaths in Virginia, we should pattern our therapy after that of Curran and our set-up after that of Bellevue. There is no place in Virginia that approaches this at present. I believe the time is near at hand when such a ward will be organized at one of our State hospitals.

Finally, it would be well for us to follow Pennsylvania, and establish a Fellowship which would allow a psychiatrist to make a special study of crime, thus becoming a penal-psychiatrist, or a penologist and a psychiatrist. A man so trained would be able to give much constructive advice in the solution of the problem of the psychopathic personality.

DR. J. S. DEJARNETTE, Staunton: I have been listening to these dictionary addresses and have heard a lot about the dictionary. As I have listened to the questions discussed this evening, Dr. Alderman is called an expert on the dictionary, and I think he should get out one and give us some new terms we can understand.

Dr. Arnold's paper in regard to the Constitutional Psychopath is a good one. I read an article four or five years ago from the Medical Society in Minnesota in which the psychiatrists kicked the word Constitutional Psychopath out of their vocabulary. The word does not mean what it implies, as in the case of the old negro when he saw an elephant, "thar's no such animal." The name, Constitutional Psychopath, implies a disease of the soul or mind. If a man has a diseased mind he is insane. If you call him a psychopath he is not insane. The word is derived from two Greek words, Psyche the soul and Patho the disease, literally meaning diseased soul. Psychopaths are not insane and should not be given a name implying insanity. They frequently go to an insane hospital, and when found not to be insane they are occasionally sent home or to the feeble-minded colony. In one case Dr. Arnold received a patient and later turned him out as not insane. He was presented to the court and the court promptly sent him to the State Farm.

Dr. Wilson says some of the doctors have been curing 60 per cent of their Constitutional Psychopaths. That is a constitutional insane person, which does not sound plausible to me, and I doubt if Dr. Wilson thinks it can be done. He quotes from someone who claims to have cured 60 per cent. I believe we should get a better

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name, something with a true meaning of what the patient has.

Many of these cases that are called Constitutional Psychopaths are children who are reared by indulgent parents and are given all sorts of liberties, not corrected, and taught obedience or the responsibility of life. Some are mild manic depressive, and some dementia praecox. Such cases are really insane and should be called insane. To rear children without proper control or teaching them obedience and self-denial is a dangerous thing.

"No dream of Childhood's early day
No storm that raged, no thought that ran,
But leaves its impress on the clay
That slowly hardens into man."

Most of us have evil thoughts, but none of us like to think about the evil things we have thought and done, but we are delighted to boast of our good deeds, etc.

As stated above, the so-called psychopaths are not insane and should not come in contact with the insane patients. The people we have been putting in this class should be diagnosed as manic depressive or dementia praecox, as the case may be. I hope we will use this expression, Constitutional Psychopath, less and less and not at all where the patient is not actually insane.

Criminals who are repeaters and shown they are not able to adjust themselves to their environment properly should be confined, not so much by the gravity of the crime, but for their tendency to repeat their crimes in which they are liable to do violence to people on whom they prey and liable to hurt themselves in their attempted crimes. Such cases should be confined for long periods on their past history. Dr. Arnold's suggestion is a good one: confine them for a good long time, sterilize them and turn them out.

DR. J. R. BLALOCK, Marion: I do not feel that home training in childhood has so much to do with the development of the psychopathic personality or the constitutional psychopath. Hubbard wrote an excellent article many years ago in which she compared the symptoms in psychoneurotic children with those in psychopathic children. The latter were self-centered, egotistical, as hard as nails, and did not profit by experience. It has been the general experience that these symptoms develop in children from good homes as well as from bad ones; that in both of these types of homes you find factors, causes of conflict, etc., giving rise to a great many personality disorders in children, to neuroses, or even subsequent psychoses.

I believe that the classification of the American Psychiatric Association dealing with psychopathic personality is as good as any. The distinction is made between psychopathic personality with psychosis on the one hand, and without psychosis, psychopathic personality on the other. Of the group without psychosis there are listed three groups: pathological sexuality, pathological emotionality, and with asocial and amoral trends. These are the three big groups of the psychopaths who are not psychotic. And as regards those who are psychotic, there are those who have psychotic episodes who may be fundamentally schizophrenic.

I believe that Henderson and Gillespie's definition is as good as there is. In a way it is about the same as Dr. Arnold's, and states that a psychopath is one who from childhood or infancy has shown certain exaggerations of personality features which have gotten him into difficulty with his environment, but who is not certifiable as insane, and who is not essentially feeble-minded; also having a tendency to develop psychotic episodes. I think this definition is as good as one can find. In 1939 Henderson published a book, "Psychopathic States" (Norton)—an excellent study. Kahn discusses psychopathic personality in terms of temperament, impulse and character, and presents a very elaborate classification, not easily understood, but well worth study.

As regards differential diagnosis, I think the problem of who is feeble-minded, and the distinction between that and one who is psychopathic is not difficult. However, simple dementia praecox presents a difficulty at times, and certain criminal types present difficulty. In the Criminal Insane Department at the Southwestern State Hospital we get a number, over twenty per cent of admissions, who are psychopaths, occasionally with psychosis, but more largely without. And of course we have the problem of reporting them back to the court. I think generally that the ordinary psychopath is reported back to the court as sane if his condition is shown as without psychosis. I have long wanted to make a survey and see what the practice is in different institutions for the criminal insane, in the matter of the legal responsibility of the certain types of the psychopaths received for observation and report.

I wish to thank Dr. Arnold for his very interesting and instructive paper.

DR. B. R. TUCKER, Richmond: I never heard Dr. Arnold read a paper that I didn't think very highly of it, and that I didn't get something out of it, something definite.

I don't know whether I have the temerity to suggest an additional criteria I have noted in connection with these people, because I have always been interested in disturbances in conduct—in myself and in others.

Every person whom I have found constitutionally psychopathic has exhibited the symptoms of romanticizing, fantasizing, and lying, and it seems to me that these are outstanding things with these people. I think really, broadly speaking, that we are dealing here with two conditions rather than with one. I think there is such a thing as a constitutional psychopath, and such a thing as an environmental psychopath. Now, in the constitutional psychopath it doesn't make any difference whether he is disciplined or not; it doesn't matter what his training has been. They come out of the best families and are well raised—many of them.

The environmental conduct individual—irregular conduct—whether he is psychopathic or not, can be cured by thorough treatment over a long-enough period of time. It seems to me we have two conditions that ought to be dealt with separately when we are dealing with individual cases. In my own small experience I have never known a real constitutional psychopath who has been cured by any method, and I'll stick to that till

somebody shows me where I am wrong. The environmental psychopaths, those who have been raised improperly—those who have had improper suggestions throughout their lives—can be cured, of course, provided time enough is given and they are under the proper influences and in charge of understanding individuals. I have two right now whose environments were splendid, and these two are just as Dr. Arnold described. They fit in with everything that Dr. Arnold said except, as I said, the ones I consider real constitutional psychopaths have exhibited the romanticizing, fantasizing and lying. The intelligence, of course, has little to do with either one. One of these cases made quite a reputation as a female newspaper reporter, though not sticking long to this—not sticking long to anything, as a matter of fact. The other one knows more about the battles of the World War—though he was nothing but a child at the time—the generals who commanded the troops, and the number of troops, than any commentator I have ever heard; and he has read more books than almost anybody I know. He reads books, but he cannot apply them properly, and his judgment is all wrong.

Now, the question whether these people are insane or not is another proposition. I think the real constitutional psychopath is a person who has in his genes and protoplasm such a strain that he should be considered insane. A person who has been raised improperly, and whose environment has led him into infractions of various kinds, need not be insane.

That is about all I have to say, except I would like to get Dr. Arnold's idea as to his experience with these people in their lying and fantasizing.

DR. P. G. HAMLIN, Newport News: I would like to ask Dr. Arnold what he feels is the measure of legal responsibility concerning these people. He spoke of sending them back to the court, and the court then sentencing them to the state road camp or something like that. I would like to ask him whether he feels that this accomplishes anything; whether it does any good.

The question of terminology I think is not important. The type was described a hundred years ago by Prichard, and later by Maudsley, and, as Dr. Blalock said, still later by Henderson in the Salmon Lectures. He (Henderson) classifies them under psychopathic states.

DR. JAMES N. WILLIAMS, Richmond: Dr. Arnold's paper is extremely interesting and timely.

This group of individuals is badly in need of further study in order that constructive plans can be made. In some institutions considerable work has been done with the electroencephalogram, and it has been found that normal waves have been present in a large percentage of these individuals.

Dr. Wilson has done considerable work along this line at the University of Virginia Hospital. I wish he would tell us something about his results at this time, if possible.

DR. ARNOLD, closing the discussion: I am not going to try to answer these questions; most of them I couldn't.

In regard to Dr. Tucker's statement, what he said about these patients is true. They do lie, and apparently for no reason at all. They lie merely to entertain you, and without any regard to fact. You will call them on one lie and they will start right off on another.

Dr. Hamlin asks about the responsibility of the court. My experience with the courts in this state is that they do hold them responsible.

SWALLOWED AIR.*

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The investigations of Wangensteen on the origin of intestinal gas in intestinal obstruction may necessitate a revision of the previous concepts of the origin of gas elsewhere in the alimentary tract. Wangensteen has shown experimentally that even in intestinal obstruction a relatively small proportion of the gaseous distention is due to gas of digestive or fermentative origin. According to his estimates, in intestinal obstruction, approximately seventy per cent of the gas present is from swallowed air, about twenty per cent from diffusion of gases from the blood stream into the intestine, and only about ten per cent from digestive or fermentative processes. He has further demonstrated that if air-swallowing is prevented by esophagostomy and closure of the distal esophagus, no appreciable distention occurs in ileal obstruction of dogs, and that the animals die of starvation rather than obstruction.

These observations indicate a new importance for air-swallowing not only in intestinal but also perhaps in gastric gaseous accumulations. Gas in the stomach may arise from several sources. First, it may theoretically occur from gaseous diffusion into the stomach from the blood stream but seems unlikely that this mechanism accounts for any appreciable amount of gas in the stomach. Secondly, it is a common belief

*Read before the Stuart Circle Hospital Clinical Club, Richmond, Va., February 12, 1941.

that gas in the stomach is formed from fermentative or digestive processes. There seems to be little to substantiate such a belief because if hydrochloric acid is present, it inhibits such processes, and if it is absent, the normal emptying time of the stomach is usually so short that there is insufficient time for gas to be formed. However, fermentation may conceivably be an important source of gas formation in achlorhydria with obstruction. It is obvious that chemical reactions between alkalis and hydrochloric acid produce gas in the stomach but in this event, the origin of the gas formation is usually obvious. Thirdly, it appears that swallowed air must account for the major portion of gas accumulated in the stomach, as it apparently does in the intestines. It is possible that there might be a regurgitation of gas into the stomach through the pylorus but there is little to indicate such a possibility. Finally, it seems that most of the gas in the alimentary canal is derived from swallowed air.

If the swallowing of air is such a major factor in alimentary gas, the question arises how does air-swallowing come about. It is well known that a small quantity of air enters the stomach with swallowing under normal circumstances. An air bubble above the level of the barium meal is to be expected in fluoroscopic study of the gastro-intestinal tract with the barium meal. There is probably always a little air in the mouth which is swallowed along with food, liquids or saliva. Furthermore, certain foods are frothy with air put into them to make them lighter and carbonated drinks contain compressed gas which is liberated in the stomach. But in addition to these, an excessive amount of air may be swallowed through faulty muscular coordination. To swallow properly, one should close the lips, suck in the cheeks, press the tongue firmly against the hard palate before the process of deglutition begins. By this method, excessive air in the mouth is expelled through the nasopharynx. Failure to coordinate in this sequence leads to the abnormal swallowing of air.

In normal individuals, proper deglutition occurs automatically and the small quantities of air ingested produce no discomfort. Loss of the knack to swallow properly may result from faulty habits such as eating too rapidly but it seems particularly prone to occur in nervous individuals. Such nervous individuals may be nervous only from some unusual nervous tension, as from illness. Many of them are confirmed psychoneurotics.

About two years ago, I examined an elderly lady who had no complaints of significance. In the routine history, it was revealed that she belched more than average but the symptom hardly required treatment. Six months ago, elsewhere she developed ascites and became excessively uncomfortable from the combination of ascites plus an inordinate amount of gas. A film of the abdomen showed the stomach distended to unbelievable proportions with gas, but two days later, when a barium meal was given, the stomach was of normal size. She then came under my care again and was proven at operation to have a papillary adeno-carcinoma of the right ovary with metastatic peritoneal implants. After following the anti-aerophagia regime, to be discussed later, she was infinitely more comfortable although the ascites continued unabated post-operatively. Apparently this patient would not have developed aerophagia of clinical importance had it not been for the superimposed strain of her organic illness.

As the result of improper swallowing an unusual amount of gas may be ingested into the stomach. This produces a sensation, often postprandially, of a slight epigastric discomfort and the subject feels that if he could just belch successfully, it would afford relief. In these circumstances, many people try to force a belch spontaneously. In order to accomplish this, usually subconsciously they suck in a little air and swallow it. If a belch ensues, the sensation may be so satisfying that the swallow-belch cycle is continued in an orgy of gaseous eructations. If, on the other hand, no eructation is produced, the intragastric tension is increased and hence the discomfort intensified. Most of the air in the stomach which is not belched up is passed on into the intestines where it contributes to intestinal flatulence as already outlined.

Some years ago, a woman in her thirties was referred because of violent generalized abdominal pain at night for the preceding six weeks. About a year previously, she had had a radical breast amputation for malignancy and had constantly feared a recurrence or a metastasis. After the abdominal pain began, she and her physician were both convinced, in spite of a negative gastro-intestinal roentgen study, that the pains were due to metastasis. In the office she was observed to swallow air copiously and was then asked if the nocturnal pains were associated with distention or relieved by the rectal expulsion of gas. When she replied affirmatively to both questions, she was placed on an anti-aerophagia regime, with complete, immediate and permanent relief. She has had no further abdominal discomfort and no return of the carcinoma.

The effect of an excessive distention of the stomach by gas leads not only to epigastric discomfort, but may also produce acute pain in the lower back, and the chest either anteriorly or posteriorly. The in-

creased size of the distended stomach may "crowd the heart and lungs," giving rise to palpitation, tachycardia, cardiac irregularities, and dyspnea. The substernal pain may even be confused with true anginal pain. A sensation of oppression in the head, dizziness, tinnitus and nervousness often seem to accompany the epigastric distention although the exact explanation for these sensations is not clear.

A few months ago, a young white mill worker was seen in the Out-Patient Clinic because of pain in the stomach of two and a half years' duration. The pains were usually dull but had been sharp at times with radiation to the lumbar region. The pain was accompanied by dizziness, "oppression in the head", nervousness and palpitation on exertion. On direct questioning, it was ascertained that belching usually relieved the symptoms and that the patient often belched for hours. Shortly after onset of the symptoms, the patient had been admitted to his local hospital where he remained for eight days without relief. Subsequently, he had had an appendectomy, tonsillectomy and hemorrhoidectomy without relief. He had been incapacitated for work by his illness and had lost twenty pounds in weight. After two weeks on an anti-aerophagia regime, he reported that he was practically well and had returned to work.

The epigastric discomfort may be extremely severe, even to the point of simulating gall bladder colic. On the other hand, it is not clear why a diseased gall bladder should be said to be a cause of flatulence, unless patients with gall bladder disease are more likely to swallow air.

Although only three articles in English have been published on aerophagia in the past ten years, and medical textbooks almost ignore the condition, it is the writer's opinion that aerophagia is the most frequently occurring and the least frequently recognized cause of digestive symptoms.

There is no condition that can be more easily recognized from the history than aerophagia, provided one remembers to ask, "Do you have an excessive amount of belching." Anyone who belches more than four or five times is presumptively an aerophagiac. Physical examination is helpful only if, as is often the case, the patient puts on an exhibition of air swallowing while in the office. Excessive distention in the epigastrium or over the whole abdomen may be suggestive but it is not conclusive. The diagnosis may be clinched by seeing air pass with the barium into the stomach during roentgenologic study.

The great pitfall in the diagnosis of aerophagia is that this symptom may obscure more serious organic disease, which must be excluded by thorough diagnostic studies in many instances, especially in older subjects. Nearly all aerophagiacs are psychoneurotics and it is often necessary to obtain a complete situational analysis in order to do the patient full justice. The importance of the psyche in this condition cannot be overemphasized.

Once the diagnosis of simple aerophagia is made, the condition can usually be most satisfactorily treated. The most difficult part of the problem often is to convince the patient of the fact that they are swallowing air and that the gas belched is not due to fermentation or their "food turning to gas." If one encounters a patient in an exhibition of the swallow-belch cycle, this can almost invariably be dramatically terminated by separating the lips so that unconcious air-swallowing is discontinued. As soon as the air already ingested has been expelled, the belching ceases and it can be explained to the patient that the "cure" was simply the result of stopping the air-swallowing.

Many years ago, I was called to the country one cold winter night because a fair, fat fortyish female was "having another gall bladder attack". This patient had been advised by doctors whom she had told about her attacks that a cholecystectomy was necessary. No doctor had previously seen her in an acute attack. On arrival, the patient was sitting bolt upright in bed, expelling such large amounts of air that she could have inflated a dirigible. Propping her lips apart with a cork relieved her pain a few minutes but this treatment was so undignified that I have not been called again. However, I happen to know that she still possesses her gall bladder.

At times, this procedure will convince the sufferer of the true cause of the discomfort and they will become cooperative to the extent of overcoming the "bad habit". Otherwise, it may be a laborious task to obtain the patient's confidence and cooperation. However, in most instances a time-consuming explanation of the mechanism of air-swallowing will convince a reasonable patient.

Recently a tuberculous young white woman was sent to the Medical Clinic for an opinion on the advisability of a thoracoplasty operation because she had lost twenty pounds in weight while a partial pneumothorax was continued. Excessive belching was observed and on questioning she volunteered that "she couldn't eat because all her food turned to gas". She had complained of the gas while at the sanatorium and had been told that she was "just nervous". No instructions had been given her about how to stop the gaseous eructations. In the clinic, we spent much time in giving her anti-aerophagia instructions and asked her to report her progress by mail.

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A week later she wrote that she had gained five pounds and was symptom-free. The thoracoplasty has been deferred.

Patients must be impressed with the fact that they have unconsciously acquired a "bad habit," comparable perhaps to "thumb-sucking," which they —and they alone—can conquer. The responsibility for victory must be placed upon the sufferer but the physician must assume the position that it is a habit that can be cured by the patient, provided he will devote a sufficient time to overcome the difficulty.

The next most important point in treatment is to tell the patient how to swallow properly. One must close the lips, suck in the cheeks, press the tongue against the roof of the mouth before swallowing whether they are taking food, liquids or are merely swallowing saliva. The physician should then demonstrate the process and insist that the patient repeat it after him. When the patient's technique is satisfactory, it must be insisted that they concentrate on the process of deglutition every time they swallow, even saliva, for a few days until they have re-educated themselves in the proper way to swallow. This is all important but it is not as simple as it sounds.

Taking air into the mouth over liquids while drinking is not uncommon but can be easily obviated by insisting that the patient always make a "water-seal" with his upper lip on the liquid which prevents the intake of air above the fluid.

A wealthy patient was seen recently complaining of indigestion, which on further questioning meant epigastric distress relieved by belching. He had been examined by several well-known physicians in New York and had spent a week in diagnostic studies at the best known middle western clinic, but his symptoms had continued. It was obvious even without the comprehensive diagnostic studies which were repeated that this patient suffered primarily from air-swallowing but in spite of all efforts on our part, his belching continued. On the day before he was to leave the hospital, he was observed drinking a glass of water. His difficulty was then apparent. He was taking in more air above the liquid than he was taking liquid. When shown how to make a "water-seal" on the glass with his upper lip, his belching ceased.

Patients must be told that they should try *not* to belch spontaneously because by so doing they subconsciously begin swallowing air in order to produce the belch. This is difficult for some people to understand but it is a point which must be emphasized. If belching is absolutely imperative, it is preferable to use soda, a carbonated drink or a carminative.

Finally, since nearly all of these sufferers are nervous, it seems worthwhile to routinely administer a sedative. A drachm of elixir of phenobarbital before meals is generally satisfactory. The medication often receives the credit from the patient in successfully treated cases, but the remainder of the regime is probably of greater importance.

An out-patient case, who had been incapacitated for two years by aerophagia, was recently seen. He had been to numerous physicians and tried numerous medicines. The diagnosis was obvious and routine instructions were given. Two weeks later, he wrote back saying that he was entirely symptom-free but that he wanted some more of that "marvellous red medicine that had cured him".

In summary, swallowed air is the most important factor in belching and probably also in intestinal flatulence. It produces a variety of symptoms and is often overlooked as a cause of indigestion. It can be easily recognized and usually successfully treated by simple measures.

PELVIMETRY AND ITS EVALUATION.*

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Pelvimetry, as its name implies, means the measurement of the diameters of the pelvis. At the outset, it may be stated that the female pelvis is of tremendous importance to the obstetric specialist and to any practitioner who assumes the responsibility of giving obstetric care to his patients. The course of every labor is influenced by the relationship between the shape of the maternal pelvis and the size of the fetal head (passage and passenger). Since anomalies of the fetal head are rare as compared with abnormalities in the size and shape of maternal pelves, and the fetal head possesses a degree of moldability which the maternal pelvis does not, the importance of careful study of the pelvis of every patient presenting herself for obstetric care becomes apparent.

*Read by invitation before the Grace Hospital Staff Meeting, January 8, 1941.

It may not be amiss, at this point, to briefly outline an historical sketch and a general survey of this subject to bring about comparative studies between former years and modern day practice.

The bony pelvis, and more particularly the deformed pelvis, received scant medical consideration to the time of Van Deventer at the turn of the eighteenth century. Vesalius, in the sixteenth century, was the first to give an accurate description of the normal pelvis. Van Deventer made no measurements of the pelvis but he recognized the variations in normal and deformed pelves. Of greater clinical significance was the work of Jean Louis Baudelocque (1789) who developed the technic of accurately measuring the normal and distorted pelvis in the living woman by means of a pair of calipers or compasses, and described the external conjugate diameter which now bears his name. Then came the pelvimeter, an invention of G. W. Stern, which placed in the hands of the obstetrician an accurate means of determining the various diameters of the pelvis. The way was now open for the exhaustive researches, both anatomical and clinical, which form the basis for our modern conception of the contracted pelvis and its relationship to childbirth. Thus, in 1851, Gustav Michaelis, professor in Kiel, and his successor, Karl Lutzman, each carefully measured the pelves of one thousand German women.

It was formerly thought that by measuring the distance between the spinous processes of the sacrum and the symphysis pubis and subtracting from it what it is judged to be the thickness of the bones and soft parts, one might arrive at an approximate estimate of the measurement of the conjugate diameter of the pelvic brim. It is now admitted that this method can never be depended on, and that, taken by itself, it is practically useless. A change in the relative length of other external measurements of the pelvis is, however, often of great value in showing the existence of deformity internally, although not in judging of its amount.

It is essential that the physician be able to diagnose the existence and extent of a pelvic contraction before the onset of labor, in order that he may, as far as possible, decide in advance upon the proper course of treatment to be carried out in each case. With this in view, accurate pelvic mensuration should constitute a regular part of the routine examination of the pregnant woman and, in keeping with our present-day knowledge, the physician who practices obstetrics should utilize pelvimetry on the same principle as the internist who treats diseases of the heart and lungs with the aid of auscultation and percussion.

At the preliminary examination, which should be made early, either on the first or second visit, the physician should neglect no points in obtaining all possible data bearing on the case. Generally speaking, large, well-built women are likely to have normal, and undersized women contracted pelves; but this rule is by no means constant and it is not unusual to disclose some abnormality in the former and perfectly normal pelves in the latter.

Several years ago the *San Francisco Examiner* stated, in large type across the page, "San Francisco Women's Hips Largest". So much the better for San Francisco and its future generation. Under this caption the famous columnist, Arthur Brisbane, said, "A woman built like a wooden lath is all right for Ziegfeld's Follies, well-built to crawl through a picket fence. The mothers of great men, from Charlemagne to Lincoln, from Alexander's mother, Olympias, who danced with snakes wrapped around her, with no other clothing, to the mother of Renan with good peasant blood, you will find real hips, something substantial, not an osteological anatomical specimen of pelvic depression".

The gait of the patient should be carefully observed since the existence of a limp or some peculiar way in which the feet are placed upon the floor may serve to direct attention to the possibility of a pelvic deformity; marked abnormalities of the spinal column—kyphosis or lordosis—are also suggestive, and even slight degrees of spinal curvature should not be overlooked as they are frequently of rachitic origin. The more usual signs of rachitis—deformities of the extremities, the characteristically shaped head and the rachitic rosary—should always be looked for. Inquiry should be made as to the age at which the patient first learned to walk and if she is found to have been backward in this respect the possibility of a rachitic pelvis should be borne in mind even though the usual external evidences of the disease may be lacking.

If the patient is a multipara she should be questioned as to the course of previous labors and the history of any serious difficulty should always suggest the possibility of an abnormal pelvis. On the other hand, a negative history is by no means valuable, as it is a well-known fact that in moderate

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degrees of pelvic contraction the first labor may be relatively easy, while each successive one becomes more difficult.

In primiparous women a markedly pendulous abdomen or the absence of engagement of the head in the last month of pregnancy should always be regarded as evidence of the existence of a marked disproportion between the child's head and the pelvis, until careful examination proves that such is not the case. This leads up to the question of pelvimetry.

While the above-mentioned conditions are of value in suggesting the possibility of pelvic deformity, accurate information as to its existence and extent can be obtained only by measuring the pelvis. For this purpose external and internal pelvimetry are employed, i. e., measurements are taken from the surface of the body and through the vagina. This includes external measurements, internal measurements and measurements of pelvic outlet.

External Measurements: Inter-spinous, intercrests, bitrochanteric, right oblique, left oblique, external conjugate (Baudelocque), and circumference of pelvis.

Internal Measurements: Conjugate diagonalis and conjugate vera.

Measurement of Pelvic Outlet: Tuber ischii, anterior sagittal, posterior sagittal, antero-posterior, depth of symphysis and estimation of pubic arch.

The Value of External Pelvimetry: In the description of the external conjugate, Baudelocque stated that by deducting three inches from it the length of the true conjugate could be accurately estimated. Later experience, however, has shown that these conclusions were incorrect and that the length of the external conjugate does not give a very clear-cut idea of that of the conjugate vera, since several other factors may exist. Thus, the amount to be deducted varies with the thickness of the sacrum and the symphysis pubis, and also depends, to a great degree, upon the elevation of the promontory of the sacrum and the length of the spinous process of the last lumbar vertebra. As a rule, when the external conjugate measures between 20 to 21 cm., the conjugate vera will rarely be found to be shortened; when, however, it measures between 18 and 19 cm., the conjugate vera is shortened in about one-half of the cases; and when it is below 17 cm. pelvic contraction is almost always present.

The external measurements are of considerable value in that they serve to indicate with a fair degree of certainty the variety of pelvis with which one has to deal. For example, the distance between the spines is 2.5 to 3 cm. less than that between the crests; but in the rachitic pelvis, owing to the flaring of the iliac spines, this proportion becomes altered, and the two measurements approximate one another in length, the former being equal to, and sometimes exceeding the latter. If both measurements are much below normal, and at the same time the external conjugate is proportionately shortened, one might conclude that the entire pelvis measures below normal in all its diameters and that a pelvic contraction exists.

These external measurements must be corroborated by internal measurements chiefly of the antero-posterior diameter, by which alone we can estimate the amount of the deformity. When the diagonal conjugate is obtained, a deduction of 1.5 to 2 cm. is made and this will give the true conjugate.

The Value of Internal Pelvimetry not only serves as a means of finding out the true status in the pelvic cavity (basin) but also as a basis for the morphologic classification of pelves. In addition, certain other informative factors can be revealed, such as palpation of the sacrum, coccyx, promontory and ischial spines. The presence of a pseudo-promontory, exostoses, and osteologic pelvic tumors may be noted.

During the process of measuring the diagonal conjugate, the anterior surface of the sacrum should be palpated from below upward and its vertical and lateral curvature noted. At the same time the mobility of the coccyx should be tested by seizing it between the fingers in the vagina and the thumb externally. In normal pelves only the last three sacral vertebrae can be felt without pushing up the perineum, whereas in markedly contracted varieties the entire anterior surface of the sacrum can be readily palpated.

The Value of Measurements of the Pelvic Outlet furnishes information as to whether this portion of the pelvis is normal or if there exists an outlet contraction. In about 5 per cent of all women there exists an outlet contraction, and probably represents the most usual type of abnormality encountered in the white women of this country, especially those in the upper classes; and, as they may give rise to serious dystocia, should receive full consideration and constitute an integral part of the routine examination of the pelvis. By palpating the pubic

arch, observations may be made whether the arch is wide (normal), narrow or angulated. When the latter occurs it approaches the male type. In the women with a normal or gynecoid pelvis, the rami of the pubis are short and the subpubic arch is wide; this makes the pelvis shallow and gives the baby a shorter trip from the superior strait to the inferior strait. The wide arch at the symphysis allows the baby in being born to hug the symphysis more closely; hence, the perineum is not so frequently stretched or torn. Said Dr. Oliver Wendell Holmes, displaying the pubic arch of a female pelvis to his medical students, "Gentlemen, this is the triumphal arch through which every candidate for immortality must pass."

SUMMARY

Pelvimetry of itself may be regarded as playing an important role in the general examination of the obstetric patient, analogous to a complete blood count or some other laboratory procedure in any given case, and serves as an aid in outlining the pelvic architecture, thus enabling one to draw certain important conclusions. As an auxiliary agent, it may be mentioned that, when disproportion is suspected in these cases, a radiographic examination may ascertain the *fetal head-pelvic diameter ratio* with a fairly high degree of accuracy.

A serious attitude toward the fundamental obstetric principle of obtaining previous knowledge of the relationship between the fetal head and the maternal pelvis (before labor actually sets in) will prevent many accidents which occur needlessly during labor, and reduce infant and maternal mortality.

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Professional Building.

THE PHYSIO-PATHOLOGY OF UNCONTROLLED DIABETES MELLITUS.

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Introduction: After scrutiny of the literature we find that there has been no paper written on this topic as such. The numerous articles referred to were all published with the intention of establishing some other point. We have attempted to drain from them vital physiologic, pathologic and clinical facts and to reconstruct them into a form which would stress the significance of the issue raised in this paper.

The Issue: Stated concisely, our issue is this. A patient has diabetes mellitus. It is satisfactorily controlled for a period of months or years. Suddenly this patient's clinical pathology runs amuck. The blood or urine shows increase or decrease of sugar—perhaps ketone bodies appear—perhaps the patient becomes ill with symptoms of hypoglycemia. The question is: what factors must be considered in determining the cause of this departure from a previously controlled state, and how may they be explained?

We do not intend to offer differential diagnoses of the various conditions nor to suggest the indicated chances in treatment—but purely to point out the factors which an alert physician must consider under such circumstances, and to substantiate our views by experimental evidence and plausible discussion of the mechanisms involved.

FACTORS UNDER PATIENT'S CONTROL: *Diet irregularities* are at once the most frequent and most obvious of all; and yet they are too often forgotten. Improper preparation of the diet prescribed, as when a new cook with different ideas of household measures comes on the scene, will cause a change in the regulation. Failure to eat a full diet will permit the therapeutic insulin to bring the sugar level below normal. We have seen diabetic children in institutions time after time deliberately upset a dish of food under the table, in the expectation that when they started to go into shock from insulin hypoglycemia they would be given candy. On the other hand, over-

eating leads to trouble more frequently than any other single cause. The patient rationalizes this by believing he is using smaller portions and can, therefore, eat more of them. Since he doesn't feel that he is over-eating, the physician must very carefully analyze every alimentary step the patient takes. Losing part of the diet through vomiting, pyloric obstruction and diarrhea will lower the glycemic level both by lack of intake and by excessive insulin action.

The advent of prescribing *exercise* for a diabetic patient in the hopes of oxidizing more sugar and requiring less insulin will affect the degree of control of the diabetes.[2] Individual ideas as to different degrees of exercise certainly vary. A patient told to do a particular amount of exercise may do much more than that. When the physician notices that the patient's blood sugar runs lower than it should with that amount of insulin, and questions the latter concerning his exercise, the patient may in good faith reply that he is doing just what he was told. Actually, unless the physician investigates this thoroughly, the patient may have to go into shock before the cause of the faulty control is detected.

Mechanical factors may turn out to be the cause; e.g., errors in filling the insulin syringe, or using wrong concentrations of insulin per cc. in the bottle as compared with the scale on the insulin syringe, will lead to gross irregularities in control. Further, using the same site of injection repeatedly delays or prevents absorption of insulin. This necessitates increasing doses, which, when injected into a new site, produces a hypoglycemia.[2]

FACTORS NOT UNDER PATIENT'S CONTROL: Another group of transient factors is the *accidental*. Fractures may go undetected and be the cause of internal hemorrhage. This leads to progressive loss of blood with resulting concentration of its chemical substances. Trauma to the skull may injure the floor of the fourth ventricle of the brain and result in glycosuria.[3] The fact that these conditions are transient does not make them any less important. Nothing is so confounding as obscurity.

We come now to a group of important and prolonged causes of failure to control the diabetic state —namely, the *endocrine disorders*. Much new light has been thrown on these variations by Anselmino and Hoffmann's observations that alkaline extracts of the pituitary cause acetonuria, and those of Houssay and Long that hypophysectomy and adrenalectomy diminish or eliminate ketosis.

Pituitary: Varied pituitary activity will effect changes in the glycemic level.[4] The eosinophilic cells of the anterior lobe elaborate a diabetogenic (or glycolytic) hormone. This renders liver glycogen less stable, so that any depression of blood sugar is rapidly countered by an outpouring of glucose from the liver, not only returning the blood sugar to normal but often carrying it to abnormal heights. It is believed that this glycolytic factor antagonizes the action of insulin in the peripheral tissues.[5] Hence, eosinophilic hyperplasia with acromegaly, or a slow-growing neoplasm, will lead to endless difficulty in regulating diabetic metabolism.

Thyroid: In regard to the thyroid, it may be said that myxedema decreases the blood sugar. Fitz and Allen believe that this is due to a depression of general metabolism rather than to reduced activity of the thyroid gland *per se*.[6] Hyperthyroidism, on the other hand, which is frequently associated with diabetes,[7] certainly must be considered as being a factor behind an increased sugar in blood or urine. Since over-function of the thyroid gland predisposes to diabetes mellitus,[7] surely a variance in the hyperthyroid state will produce a change in the course of the carbohydrate metabolism—and subsequently fat combustion. This gains importance when we appreciate that many cases of mild thyroid disease go undetected. The most prevalent belief today is that the thyroid factor acts by way of the liver, inducing glycogenolysis.

Adrenal Cortex Disturbance is a third endocrine factor defying control of the diabetic state. Although the mechanism is not completely understood, it is believed that the cortical hormone is concerned specifically with carbohydrate metabolism, since depletion of liver glycogen and hypoglycemia occur in cortical insufficiency (Addison's disease) and are prevented by cortical extracts. Silvette and Britton[8] have reported that liver glycogen and blood sugar are raised above normal values by injection of the extract.

Menstruation: Physicians who had been treating diabetic children observed that there came a time when the usual regime did not suffice. This was particularly true in growing girls who, for a period of a year or so, seemed to require increased amounts of insulin. Further studies revealed the relationship of increased blood sugar to the onset of menstruation.[2] In all probability this is caused by the increased amounts of diabetogenic hormone produced

by the anterior pituitary which at this period of life is more active. The excess insulin required can usually be decreased somewhat a year or two after the establishment of regular menstruation.

Pregnancy: The relationship between diabetes and pregnancy has for a long time been known to be important. From our point of view, it is particularly significant early in pregnancy, when the condition is not suspected, and yet severe metabolic shock is imposed on the diabetic state. The physician may fail to become aware of this complication either because he does not suspect it and it is not offered voluntarily by the patient, or because the patient, if asked, may for reasons of her own deny it, or, as is frequently the case, the patient herself is really unaware. Wilder[9] and others claim that pregnancy may aggravate an existing diabetes due to the ingestion of large amounts of food. On the other hand, the work of Stander and Peckham would indicate that that tendency to hyperglycemia may decrease or disappear in pregnancy. They imply that this may be due to a pancreatic hormone coming from the fetus, or perhaps a greater demand by the fetus for maternal carbohydrates. Their conclusions are based on careful studies of urine and blood sugar, ketone bodies and ketogenic-antiketogenic ratios.[10] These two antagonistic views serve to emphasize the importance of the problem. No matter what the cause, and no matter what the effect, pregnancy distinctly alters the otherwise uneventful control of the diabetic patient. To generalize, it may be said that the first semester of pregnancy tends to aggravate the diabetes as evidenced by increasing hyperglycemia and glycosuria, while the latter half of gestation lowers the blood and urinary sugar. In either case, however, the problem of control is made more difficult. With methods for earlier diagnosis of pregnancy, the alert diabetes clinician will detect the gravid state in time to calculate for the factors in variance and reestablish control.

Acute Disease: Along the line of acute disease, suffice to say that fever is accompanied by acidosis. All infectious phenomena obscured by *insidious onset* must be looked for. *Carbuncles,* though upsetting the diabetic equilibrium severely, are externally manifest and should offer no difficulty in being detected as the cause of a rising blood sugar and the presence of ketone bodies in blood and urine.

Trichomonads: Routine observations on the quantity of sugar in the urine of a diabetic who also harbored Trichomonas hominis in his intestine, led to the impression that glycosuria and the number of Trichomonads in the stool tended to increase and diminish at the same time.[11] This is explained by the novel concept that the intestine, as well as the kidney, may act as an avenue for release of sugar in hyperglycemic states. If this were true, the amount of carbohydrate in the intestinal contents would increase as it does within the urine. This is borne out by the fact that the metabolic and reproductive activities of other parasitic forms (Entameba histolytica, Balantidium coli, Trichomonas fetus, *et al*) are stimulated by the presence of carbohydrates. Further, Trichomonas hominis itself is known to increase in greater numbers *in vitro* in the presence of added carbohydrate.[12] When this is the case, gastrointestinal disurbances may occur, with diarrhea, vomiting, hemorrhage, anorexia, etc., all of which obviously lead to difficulties in controlling the diabetic state.

Chronic Disease: In the presence of chronic infection, ability to utilize carbohydrate is impaired. One should, therefore, thoroughly search for chronic sinus or middle ear infection, alveolar abscesses, chronic pulmonary conditions, chronic infection of the gall-bladder, appendix, Fallopian tubes and prostate gland.

Pulmonary Tuberculosis requires special comment because it is now known that it occurs more frequently among diabetics than non-diabetics.[13] The problem arose from the fact that while treating the diabetes the patient became sicker and sicker from the tuberculosis, since his resistance was not allowed a chance to improve. Treating one, meant decreasing the diet; treating the other, meant increasing it. This has been overcome by the advent of insulin; yet, since it is not uncommon for diabetic patients with tuberculosis to develop ketosis,[14] one should suspect the presence of tuberculosis in that circumstance.

Syphilis bears relation to diabetes by way of producing a specific lesion in the brain.[15] Syphilitic arterial disease with secondary nervous lesions in the region of the fourth ventricle, or gumma in this part, will produce hyperglycemia and glycosuria. Again, this might be due to syphilitic disease of the blood vessels of the pancreas and secondary disease of pancreatic gland tissue. Rosenbloom's review of the literature on this relationship fully corroborates this view.[15] This does not mean that lues causes diabetes, but does imply that certain cases of syphilitic

pancreatitis may be severe enough or peculiar enough to produce the blood, urinary and metabolic findings characteristic of the diabetic state. Since syphilis often goes unadmitted or undetected until tertiary signs are manifested, it is essential to keep this possibility in mind where difficulties arise in maintaining the desired glycemic level in the patient.

Liver: The role of liver pathology in uncontrolled diabetes has become increasingly important. A diabetic state *per se* has never been known to cause cirrhosis. It does, however, lower the metabolic integrity of the liver,[16] and among other things brings down the glycogen content to a subnormal quantity. This, together with a chronic alcoholism and an added irritant toxic factor, will produce a cirrhosis;[17] and thus, with less glycogen storage, a hyperglycemia results. The sequence of events appears to be fatty infiltration, hyaline degeneration, atrophy of the cells at the periphery of the lobules, and fibroblastic proliferation, ending in the typical fibrotic structure of cirrhosis. This has been shown to occur in depancreatized dogs maintained with insulin.[18, 19] The significance of this lies in the fact that liver changes make control of diabetes difficult by virtue of aiding in the maintenance of a more or less chronic acidosis. More insulin will be required for these patients. The relationship alluded to is borne out by the observations of Hart and Lisa[17] that cirrhotic ascites in a diabetic disappeared when insulin dosage was increased.

Leukemia: There is an interesting series of five cases reported wherein diabetes was treated successfully until a certain date, when everything went wrong. It was not controlled again until the diagnosis of leukemia was established, and radiation therapy instituted to control the leukemia. Post-mortem examination was performed in only one of these cases and leukemic infiltration was detected in the pancreas. Had the other cases been autopsied, they might have substantiated this view of the effect of leukemia on the control of the diabetic state. Four of these five cases were the myeloid type.[20] This relationship has come to be of rather frequent occurrence.

Psychic, Nervous and Emotional Factors have gained importance since Le Winn reported a hyperinsulinism improved during pregnancy by psychic trauma.[21] Severe nervous shocks such as may result from injuries, exposures, anxieties, fears, unhappiness from domestic infelicities and financial losses, are notorious factors in maintaining glycosuria. Various injuries and diseases of the central nervous system, such as concussions, skull fractures, apoplexies, brain tumors, etc., act in the same way. It is true that such glycosuria is often transient rather than diabetic; but if, as Woodyatt[22] claims, such factors can actually provoke the onset of a true diabetes previously non-existent, certainly they can prove very troublesome in the course of existing diabetic states.

Insulin Resistance and Sensitivity: Insulin resistance may be defined as the requisition by a patient of more than 100 units per day. The importance of this factor in uncontrolled diabetes is seen as soon as it is appreciated that extreme cases of ketosis may require more than 3,000 units in twenty-four hours to be effective.[23] Somogyi[24] has postulated that the explanation of insulin resistance lies in the following: large doses of insulin reduce the blood sugar; this low glycemic level stimulates the suprarenal glands to produce an excess of epinephrine; this raises the blood sugar, and constantly counteracts the action of insulin.

Himsworth[25] and Nadler[26] independently have shown that insulin sensitivity is due to lack of insulin, and that carbohydrate increases sensitivity. They believe that the effects of insulin are governed by an unknown factor or condition, a deficiency of which results in impaired peripheral action and is responsible for insensitiveness or resistance to insulin. That the liver cannot be solely responsible is indicated by the repeated finding that sensitiveness to insulin induced in the normal dog is not abolished by hepatectomy.

Since a patient on a high carbohydrate diet is likely to become relatively insulin resistent, and a patient on a low carbohydrate diet is likely to become insulin sensitive, this factor should be appreciated when changes in blood and urinary sugar and sugar tolerance curves turn up in a previously regulated individual. The development of high carbohydrate tolerance and insulin resistance appear to have been aided by the use of protamine zinc insulin.

It should be added that allergic reactions, Trichomonad infestation and tuberculosis have been reported as factors associated with insulin resistance.[26]

Miscellaneous Factors: Finally, such miscellaneous factors as prolonged exposure to sunlight, high altitudes, low fluid intake and dehydration from other causes, and acid-ash diets must be considered.

These render less insulin necessary and hence favor hypoglycemic reaction if the usual dosage is maintained.

SUMMARY

We have stated that many cases of regulated diabetes become transiently or permanently uncontrolled. No inclusive paper on the varied causes of this phenomenon has been discovered. We have attempted to gather these causes together and, where possible, to explain the physio-pathology involved. The related conditions may be classified as follows:

A. Factors under Patient's Control—
   1. Diet Irregularities
   2. Variance of Exercise
   3. Mechanical Factors.

B. Factors not under Patient's Control—
   1. Accidental—trauma, hemorrhage, piqure
   2. Endocrine Disturbances
      a—Pituitary
      b—Thyroid
      c—Adrenal Cortex
      d—Menstruation
      e—Pregnancy
   3. Acute Disease
      a—Insidious Onset
      b—Carbuncle
      c—Trichomonad Infestation.
   4. Chronic Disease
      a—Visceral Infection
      b—Tuberculosis
      c—Syphilis
      d—Liver Disease
      e—Leukemia.
   5. Psychic, Nervous and Emotional Factors
   6. Insulin Resistance and Sensitivity
   7. Miscellaneous Factors.

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LEUKOPENIA—IMPRESSIONS AND REVIEW OF CASE REPORTS.*

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In most textbooks of medicine it is stated that individuals in whose blood there are found 5,000 leucocytes or less per cubic millimeter show a state of leukopenia. There are few data available concerned directly with the study of leukopenia. Nevertheless, Roberts and Kracke, in a statistical study of 8,000 records of patients seen in private practice for the ten year period be-

*Read before the Clinical Club of Stuart Circle Hospital, Richmond, Va., March 12, 1941.

tween 1920 and 1930, recorded that leukopenia occurred in 23 per cent of their patients. Such a percentage is impressive enough to indicate that the paucity of material on this subject must be due to frequent failure of the physician to perceive the condition rather than to the infrequency of its occurrence.

During the past few years I have encountered a number of cases of mild leukopenia with vague symptoms of chronic fatigue, lassitude, lack of appetite, lack of endurance, and often nervousness, which symptoms could not be based upon any physical findings, dietary deficiency or lack of proper living habits on the part of the patient. So much has recently been written regarding agranulocytosis following toxicity from drugs, especially the benzine group, which causes an aplasia of the bone marrow and lack of maturation of the white cells, that I will not mention further this type of leukopenia. However, only since 1922, when agranulocytosis was first reported by Schultz, has a true significance of the leukopenic state been fully appreciated. Before that time white blood cell decrease was sometimes regarded as a valuable diagnostic sign, but it was not recognized that wholesale bacterial invasion might soon follow this decrease in the number of protecting leucocytes.[1]

It must be borne in mind that each granulocyte lives only a short time, probably not over 4 days, so that many cells have to be released from the bone marrow every day to make up for those normally destroyed. Apparently the granulocytes have little function while in circulation.

The factors involving leukopenia are:

1. The activity of the bone marrow—the bone marrow may be aplastic or hyperplastic.
2. Increased elimination of white cells.
3. Increased rate of destruction.
4. Abnormal distribution of white cells.

Among the factors which affect granulopoiesis are: infection; chemicals; radiation; excessive sunlight, which depresses bone marrow to a variable extent in some people; diet; hormones; physiological failure in the aged and often in people who are not aged; aplastic anemia; aleukemic leukemia; pernicious anemia during periods of relapse; Banti's disease, and various infections, particularly the virus infections, typhoid and malaria and overwhelming infections.[2]

Also, a severe leukopenia may occur in certain conditions characterized by hemorrhagic tendencies.

The following case is a rather interesting one in that it developed in a patient with acute supra-renal failure:

CASE 1—White woman, aged 40, admitted to Stuart Circle Hospital on March 28, 1937, because of generalized aching. About six months prior to her admission to the hospital, she began to have occasional spells of nausea and vomiting associated with some epigastric discomfort and slight mental depression. Her past history was irrelevant except that she had had a cesarean section twelve years ago.

When admitted to the hospital, her white blood count was 4,900, neutrophiles 61 per cent, lymphocytes 38 per cent, endothelial leukocytes 1 per cent. Red blood count 4,500,000. Hemoglobin 90 per cent.

All sorts of examinations were made, including gastro-intestinal X-ray and multiple agglutination tests, but nothing especially significant was found. Her white count dropped at one time to as low as 1,000 plus. During her course in the hospital, she consistently ran a temperature between 98.6 degrees and 101 degrees, with correspondingly increased pulse and respiration. Her blood pressure remained almost constant between systolic 70 and 80, diastolic 50 and 60, with one rise of blood pressure to systolic 140 and diastolic 108. A tentative diagnosis of acute supra-renal failure was made because of the fluctuating low blood pressure.

Her nausea and vomiting continued in spite of dietary restriction, intravenous glucose and transfusions. She was given Pentnucleotide, cortin, eschatin, liver extract, yellow bone marrow and finally X-ray treatment of her long bones, at which time her leucocyte count rose to 13,000 with 85 per cent neutrophils, gradually dropping again to an average of 4,500. She finally became comatose and died.

An autopsy[3] was done with the exception that the bone marrow was not examined. Nothing significant was found except the following: The liver

1. Kracke and Garver: Diseases of the Blood and Atlas of Hematology, Section III, Chap. XII, Page 135.

2. Kracke and Garver: Diseases of the Blood and Atlas of Hematology, Section III, Chap. XII.

3. Autopsy performed by Dr. Regena Beck at Stuart Circle Hospital.

on gross section showed moderate congestion and microscopic study showed moderate parenchymatous degeneration and moderate congestion. The supra-renal glands appeared as shells. The adrenals were found to be quite friable, each being accidently torn. They were found to be hollow organs, the cavity being lined by a dark brown friable substance which has the appearance grossly of being very old blood. They were 5 cm. in length, 2.5 cm. in width at the widest part. The adrenal substance was 5 mm. thick at its thickest part.

Microscopic study—the cells of the cortex seemed to be normal. The medulla was missing.

It was thought probable that the Leukopenia in this case was due to constitutional inactivity of the bone marrow and also to lack of proper distribution of white cells from the bone marrow because of supra-renal failure.

CASE 2—White woman, aged 33, first came under observation March 17, 1939, complaining of generalized joint pains, insomnia, acute pain and a "bluish" spot in the calf of her right leg. Her general physical examination was essentially normal except that there was tenderness and the appearance of a purpuric area about the size of a dime over a vein in the calf of the right leg. Her white blood count was 2,538, differential neutrophiles 51 per cent, lymphocytes 37 per cent, eosinophiles 2 per cent, endothelial leukocytes 10 per cent. Hemoglobin 91 per cent, red blood count normal, and temperature of 102 degrees. She was moderately obese. Her tonsils, appendix and uterus had been removed within recent years.

The leg was splinted, elevated and ice kept applied at intervals to the tender areas and she was given absolute bed rest for a period of three weeks.

Since this acute illness she has had from time to time what was thought to be a migratory phlebitis in the veins of the right arm, left arm, left thigh and right thigh, and during the acute exacerbations her white count always drops below 5,000.

When given liver extract intramuscularly, each time there has been a very definite rise of her white count until at times it has reached 9,100. She has been given liver extract at weekly intervals at her own request because of symptomatic relief. In spite of the fact that her blood cultures have been negative, her sedimentation rate at times has been elevated about twice above normal. Her past history with the exception of surgical procedures was normal. She has two children living and well—the youngest 4½ years old. She is able to perform her duties as a housewife so long as she takes liver extract.

It is thought that the leukopenia in this case is due to suppression of the bone marrow by an infection, the etiological organism of which has not been determined.

CASE 3—Young girl, aged 11, weight 94 pounds, past history irrelevant except for the usual childhood diseases and tonsillectomy. Hemoglobin 65 per cent, red count 3,000,000, white blood count 2,850, differential; lymphocytes 26 per cent, neutrophils 69 per cent, metamyelocytes 4 per cent, endothelial leucocytes 5 per cent. General physical examination normal except for a pharyngitis. Chief complaint stiffness in neck and pain in shoulders.

She was given daily injections of liver extract until her white count reached 6,700; her temperature was 101 degrees F. She recovered after two weeks rest in bed with a normal white count. In order to keep her white blood count above 5,000, it seemed necessary to continue the liver extract at weekly intervals for a month, since in from five to seven days after each injection her white count would drop below 5,000.

In both cases 2 and 3 it is thought that the low white count was due to some type of infection (which we were not able to determine by blood cultures or multiple agglutination tests), which had a suppressive effect upon the bone marrow.

CASE 4—White woman, aged 60, who had been under observation since May, 1926, at which time it was discovered that she had an acute cholecystitis with cholelithiasis and mild diabetes. She had a cholecystectomy without complications in 1926. In August, 1940, she appeared to have an acute pulmonary edema, from which she recovered after administration of concentrated glucose and adrenalin, but it was found that her hemoglobin was 37 per cent, red count 1,895,000, white count 3,400, and she since has been given many transfusions of citrated blood. Differential, lymphocytes 51 per cent, monocytes 3 per cent, eosinophiles 3 per cent, and basophiles 6 per cent.

So long as her hemoglobin could be kept above 50 per cent, which had to be done by transfusions of blood (since there was no response to liver and iron), she was able to do her usual duties as a housekeeper.

XUM

Her blood sugar was able to be controlled with the use of 20 units of insulin per day and moderately restricted carbohydrates. From repeated studies of her blood and a biopsy of the bone marrow, which was obtained by a sternal puncture, it was thought that she had aleukemic leukemia or chronic myelogenous leukemia.

On January 19, 1941, she entered the hospital with hemoglobin of less than 25 per cent, with much pulmonary edema, and was given a transfusion to which she did not respond and died.

The autopsy[4] showed an apparent aplastic bone marrow with chronic passive congestion of all organs, spleen much enlarged, fluid in the peritoneal cavity, both pleural cavities and much pulmonary edema.

CASE 5—White woman, aged 58, was seen first on March 20, 1935, because of lack of endurance, rather marked dyspnea and diffuse bodily aches. Her general physical examination, including electrocardiographic studies, basal metabolism, gastric analysis, the routine blood chemistries and urinary studies, appeared normal. Her hemoglobin was 93 per cent, red count 4,500,000, white count 3,600, differential, lymphocytes 39 per cent, neutrophiles 50 per cent, eosinophiles 5 per cent, basophiles 1 per cent, and metamyelocytes 5 per cent. Complete allergic tests were made in her case and found to be negative.

She had been given liver extract by mouth and by injection since first seen. Occasionally, however, she will omit the liver extract for a period of time and after a few weeks the symptoms recurrently appear. At the present time she is in excellent health although necessarily convinced that liver extract is essential for the continuation of her good feelings. All medication has been eliminated except liver extract.

This case seems to be one in which the leukopoietic tissues have been inactive but are kept active by liver extract.

CASE 6—White boy, aged 6, admitted to Stuart Circle Hospital on February 9, 1939. The patient had a definite spasm of the muscles of the neck with exaggerated knee jerks, an acute pharyngitis, temperature of 103½ degrees, pulse 130. There was a positive König. His spinal fluid appeared normal and there was no increase in spinal fluid pressure. The white blood count was 12,200, neutrophiles 88 per cent, lymphocytes 12 per cent.

He was immediately given sulphanilimide, tablets 2 every four hours. Within 24 hours his white blood count had dropped to 3,275, neutrophiles 75 per cent, filamented 44 per cent, nonfilamented 31 per cent; lymphocytes 21 per cent, endothelial leucocytes 3 per cent, unclassified 1 per cent.

Daily blood counts were made for a period of 8 days. The sulphanilimide was discontinued and his blood count returned to normal within 48 hours. The meningismus cleared up within a period of 10 days; also the pharyngitis.

This case was thought to be one of neutropenia due to sulphanilimide sensitivity, although, of course, it is possible that the child had a meningismus secondary to an influenza with neutropenia, which was not affected by sulphanilimide but by the type of infection.

CASE 7—White male, aged 47, first seen May 1, 1938, complaining of faintness when up and around, nervousness and some pain in the precordial region, especially upon exertion.

His routine examinations were normal except that his hemoglobin was 69 per cent, red count 2,300,000, white count 3,600. Color index 1.5, differential; lymphocytes 52 per cent, neutrophiles 46 per cent, eosinophiles 2 per cent. The gastric analysis has repeatedly shown an achylia.

He has been totally relieved by a high protein diet and his blood counts are now as follows: Hemoglobin 80 per cent, erythrocytes 4,250,000, leucocytes 6,300.

This case seems to have been one of a leukopenia due to pernicious anemia and is easily controlled by liver extract and diet.

These few reports were chosen from a series of cases carefully followed over a period of time because they represent the various types of leukopenia associated with or without a general suppression of the bone marrow as indicated by the frequent normal hemoglobin and red cell counts. Seven cases of leukopenia have been reported, five of which were of obscure origin. While it is hard clinically to divide the idiopathic or obscure leukopenia and the well-defined groups because of the obscurity of the etiological factors concerned, it is to be borne in mind that there are cases of severe leukopenia that exist over a period of months or even years causing disability without fatality.

The condition appears to be an intrinsic abnormality of hematopoiesis. As suggested by Rosenthal, among others, there may be leukopenic predisposi-

4. Autopsy performed by Dr. Regena Beck at Stuart Circle Hospital.

tion or trends in certain individuals. As pointed out by Cabot, Clough, Roberts and Kracke, a diagnosis is often made of neurasthenia, psychosis, hysteria and the various neuroses.

Out of the 10,000 case records studied by Mettier and Olsan of the University of California Medical School, the following table is of interest:

Number of Records Examined	10,000
Number with Leucopenia	1,167
Females with Leucopenia	611—52.4% of cases
Males with Leucopenia	556—47.6% of cases

CONDITION IN WHICH LEUCOPENIA OCCURRED	NUMBER OF CASES WITH LEUCOPENIA	PER CENT INCIDENCE OF LEUCOPENIA IN THE VARIOUS CONDITIONS
I		75 to 100% incidence
Influenza	137	100% of cases
Typhoid fever	14	100% " "
Brucelliasis (undulant fever)	7	100% "
Banti's disease (splenic anemia)	19	100% " "
Aleukemic leukemia	9	100% " "
Aplastic anemia	5	100% " "
"Agranulocytic" angina	2	100% " "
Arsphenamine intoxication	2	100% " "
Pernicious anemia, in relapse	109	97% " "
Malaria	29	82% " "
Myxedema untreated	30	75% " "
II		25 to 75% incidence
Sprue	4	57% of cases
Hodgkin's disease	23	52% " "
Arthritis, acute infectious	6	33% " "
Jaundice, catarrhal	9	31% " "
Cirrhosis of liver	22	26% " "
Endocarditis, subacute bacterial	13	25% " "
Lymphosarcoma	6	25% " "
III		4 to 25% incidence
Septicemia, staphylococcus	2	22% of cases
Hyperplasia of thyroid gland	54	22% " "
Hemolytic jaundice, acquired	3	11% " "
Tuberculosis (all forms)	57	21% " "
Cholecystitis, chronic	60	17% " "
Lead poisoning	3	13% " "
Rheumatic valvulitis, chronic	18	12% " "
Infectious mononucleosis	1	15% " "
Arthritis, chronic infectious	9	10% " "
Peptic ulcer	34	10% " "
Lues (tertiary)	41	9.7% " "
Arthritis, chronic hypertrophic	20	7.6% " "
Diabetes mellitus	36	7.4% " "
Pellagra	1	4% " "

Margins in medicine are indeed small, and seemingly insignificant deviations quite often prove to be of profound importance when carefully studied. It becomes increasingly obvious to the physician that nothing ever must be taken for granted. Above all else, being father confessor, family friend, protector and even priest, he must essentially be of a scientific mind. Each patient is a complete and separate entity, presenting a problem and a challenge which in the light of analytical study will reveal inevitably at least one unsuspected finding.

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Medical Arts Building.

COMPOUND FRACTURES.*

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In the last two years there has been increasing interest in the treatment of compound fractures. Trueta, in an excellent monograph, reported his results on what is perhaps the largest series of cases on record; i. e., over 5,000 cases of war injuries treated during the recent Spanish Insurrection. A host of other excellent men from various parts of the country have also written enlightening papers on this subject.

As before, there still seems to be two schools of thought. One in favor of the open treatment; the other, advocating the closed, and despite marked progress in various aspects of the treatment, the two schools cannot agree.

*Read before the Albemarle County Medical Society.

Recently the Orthopedic Department of the University of Virginia Hospital published their results of the treatment of over 250 cases of compound fractures over a seven-year period.[1] In the first three years of that period one-half of the total cases were treated by set method which included the use of iodine and alcohol in the wound. In the remaining period, the same method prevailed, except that we substituted saline for the iodine and alcohol.

It is interesting to note the method we use agrees with those used in most of the other clinics except for one or two details. Wounds treated during the period up to eight hours after the occurrence were, if possible, closed. Those seen after eight hours were, with a few exceptions, treated by the open method.

Our results were tabluated and compared to the statistics accumulated elsewhere. We were surprised to find that our percentage of infections was less than most places, while our percentage of primary healing was better than most of the clinics using the open method. Our incidence of gas gangrene was likewise low despite the fact that this section of the country is notorious for the high rate of gas infection. In 1934 Stone and Holsinger[2] reported an incidence of 12.3 per cent of gas infection in a series of compound fractures seen here over a twelve-year period. Our incidence was 3.6 per cent.

Our results are not offered boastfully. We merely wish to refute any attempt at dogmatism. We rather hope to further cut our incidence of infection and gas involvement by the local use of sulfanilamide.

(A) INFECTIONS IN CASES SEEN DURING EIGHT HOUR PERIOD

Infection	Tetanus	Gas	Non-Union	Delayed Union	Primary Healing
4.6	4.90	3.6	1.8	3.70	59%

Inasmuch as our cases are handled by an ever-changing house staff, we attempt to follow a set routine.

On entrance into the hospital, the patient's clothes are removed and a thorough examination is done. Shock is immediately combated by the application of splints to the injured area, use of the shock position, warm blankets, morphine and fluids when indicated. X-rays, though not always necessary, are taken because of medico-legal reasons. A portable unit is used, if the patient cannot be moved.

When the patient is out of shock, he is taken to the Operating Room, where the wound is covered with a sterile dressing. Anesthesia, either spinal or general, is administered. The extremity is shaved; the skin about the wound scrubbed well with soap and water for about ten minutes. Care is taken to keep the wound covered during the former procedures. The sterile bandage is then removed from the wound and the latter is gently washed with sterile warm water. Gross dirt and clots are picked out. The skin is then painted with iodine and alcohol, care being taken not to get any into the open wound.

The extremity is draped, following the usual operating room procedures. Two sets of instruments are used; the first set for the preliminary debridement and the second for the closure.

Debridement is now begun, contused skin edges and all devitalized tissue are removed. All foreign material and clots are picked out and crushed muscle is removed by sharp dissection. Bone that is attached to the periosteum is not removed. It is important to see that the recesses of the wound are opened as these areas are breeding grounds for the gas bacillus as well as other organisms.

The wound is then irrigated with two to four quarts of sterile normal saline. We use an ordinary infusion outfit, being careful to keep the bottle just above table level. This precludes developing a fluid pressure great enough to wash dirt back into the muscle planes. The saline is used only for its mechanical flushing effect.

We now discard the dirty instruments, change the drapes, and use clean gloves and gowns. Sterile sulfanilamide crystals are placed all over the wound and between the bone fragments. Care is taken not to allow the powder to collect in any great quantity at any one place as there is a tendency for this to excite a foreign body reaction.

The extremity is manipulated and, if necessary, and only if necessary, metal fixation is used. If the wound can be closed without tension, a minimum of suture material is used for this purpose. No drains are placed. A snug, lightly padded cast is applied.

The patient is given prophylactic gas and tetanus serum and sulfanilamide orally. We have also been using x-ray therapy as a prophylactic agent against the gas bacillus.

All cases are hospitalized and watched carefully.

In a recent issue of *The Journal of the A. M. A.*, Thompson[3] wrote his Ten Commandments for the Treatment of Compound Fractures. One was to

leave all wounds open and another was to use a 2 per cent aqueous solution of pectin in the wound. Orr, in another paper, absolutely forbid the use of suture material in or about the compound wound.

On the other side of the fence, Jensen[4] and his co-workers report 4 per cent infection in a series of forty-six cases treated by the closed method. They had no gas infection. Jackson[5], using sulfanilamide in fifty-four cases had 5.6 per cent infection and likewise no gas infection. Venable of Texas, Key of St. Louis, Koch of Chicago, and many others report very excellent results with the closed method. One cannot just disregard these reports.

In answer to those who refer to Trueta's large series of cases, it must be remembered that most of his cases were war injuries caused by high explosive shells and aerial bombs. In these injuries great areas of tissue were torn away and primary closure was impossible. Time also was a highly important factor. Civil injuries on the other hand are usually of two types, fractures from within with little or no loss of tissue and fractures from without, which often do carry away tissue. The latter are, in a majority of cases, probably best treated by the open method.

1. In conclusion, I feel that primary closure with the use of local sulfanilamide in a large number of compound fractures is the procedure of choice. Anybody can leave a wound open. One should not be confused by comparing war injuries to civil injuries.

2. Sulfanilamide is only an adjunct, not a means in itself, in treating these cases. We[6] have proven experimentally that sulfanilamide, when placed in a wound in which devitalized tissue is left, has practically no effect.

3. We[7] have experimentally been able to grow the Staphylococcus in a broth culture saturated with sulfanilamide. For this reason we prefer the sterile ampules of powder.

4. Care must be taken not to mistake bacteriological gas infection for clinical gas infection. The former is usually harmless.

5. Debridement is probably the most important step in the treatment of compound fractures.

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Military and Naval Section

The following have been added to the list of

Examining Physicians on Local Boards

Dr. J. L. Alexander, Staunton.
Dr. James P. Baker, Richmond.
Dr. Thomas Beath, Richmond.
Dr. James F. Blades, Richmond.
Dr. A. Gilbert Blakey, Goochland.
Dr. E. P. Bray, Jr., Richmond.
Dr. Ashby Coleman, Bastian.
Dr. A. C. Davis (Col.), Hampton.
Dr. J. L. Davis, Waynesboro.
Dr. Percy Whilt Dreifus, Yorktown.
Dr. E. M. Ellerson, Waynesboro.
Dr. Francis P. Floyd, Waynesboro.
Dr. Charles C. Freed, Waynesboro.
Dr. A. B. Gathright, Richmond.
Dr. M. T. Garrett (Col.), Waynesboro.
Dr. James T. Gill, Richmond.
Dr. J. M. Habel, Suffolk.
Dr. H. T. Hawkins, Waynesboro.
Dr. Richard S. Herring, Richmond.
Dr. Oscar L. Hite, Richmond.
Dr. J. F. Hubbard, Waynesboro.
Dr. J. G. Jantz, Bedford.
Dr. C. Kirtner Johnson, Staunton.
Dr. G. F. Johnson, Charlottesville.
Dr. Lawrence B. Kelleher, Richmond.
Dr. E. M. LaPrade, Richmond.
Dr. Kinloch Nelson, Richmond.
Dr. Dan O. Nichols, Charlottesville.
Dr. M. E. Owens, Richmond.
Dr. Barney Plotnick, Richmond.
Dr. Wm. B. Porter, Richmond.

Dr. E. A. Powell, Madison.
Dr. Thos. G. Pretlow, Chester.
Dr. Theron R. Rolston, New Hope.
Dr. J. T. Rountree, Woodstock.
Dr. Albert Russo, Salem.
Dr. M. P. Rucker, Bedford.
Dr. E. W. Stratton, Jr. (Col.), Charlottesville.
Dr. M. C. Stuart, Jr., Winchester.
Dr. H. St. Geo. Tucker, Richmond.
Dr. V. A. Turner, Staunton.
Dr. Harry Walker, Richmond.
Dr. D. Edward Watkins, Jr., Waynesboro.
Dr. B. K. Weems, Waynesboro.
Dr. J. P. West, Bedford.
Dr. George W. White (Col.), Richmond.
Dr. E. B. J. Whitmore, Jr., Staunton.
Dr. W. S. Whitmore, Staunton.
Dr. Robert H. Wright, Jr., Phoebus.
Dr. Thomas P. Davis, Jr. (Col.), Hampton.
Dr. E. Y. Lovelace, Jr., Bedford.
Dr. Chester W. Powell, Waynesboro.
Dr. Edwin M. Pilcher, Chester.

Medical Reserve Officers

In addition to those previously reported through the MONTHLY, the following doctors have been ordered to extended active duty by the War Department, Washington:

Lieut. Robert W. McCullough, Charlottesville.
Lieut. Gilman Rackley Tyler, Richmond.
Lieut. James O. Burke, Richmond.

The following have been ordered to extended active duty by the commanding general of the Third Corps Area:

Capt. Henry Louis Bastien, Arlington—Ft. Belvoir.
Capt. Herman H. Hines, State Farm—Ft. George G. Meade, Md.
Capt. Earle C. Gates, Chesterfield—Indiantown Gap Military Reservation, Pa.
Lieut. George Cooper, Jr., University—Ft. Eustis.
Lieut. William H. Kaufman, Charlottesville—Ft. Eustis.
Lieut. George E. Snider, Richmond—Camp Lee.
Lieut. Daniel Coleman Booker, Richmond—Ft. Eustis.
Lieut. Charles H. Dow, Chilhowie—Ft. George G. Meade, Md.
Lieut. George S. Fultz, Butterworth—Camp Lee.
Lieut. Brainard E. Hines, Richmond—Ft. George G. Meade, Md.
Lieut. Robert P. Meyers, Richmond—Ft. Story.
Lieut. H. Grant Preston, Harrisonburg—Ft. Story.
Lieut. H. M. Dalton, Charlottesville—Indiantown Gap Military Reservation, Pa.

Promotions

Major Richard A. Bowen, Richmond, has been promoted to Lieutenant-Colonel. He is on duty with the Medical Corps of the 176th Infantry Regiment at Fort George G. Meade, Md.

Orders Revoked

Capt. John N. Dunn, Blackstone.
Capt. Henry G. Steinmetz, Arlington.
Lieut. Manfred Call, III, Richmond.
Lieut. George S. Fultz, Jr., Butterworth.
Lieut. Edward A. Mitchell, Clinchco.
Lieut. George E. Snider, Richmond.
Lieut. Wm. D. Chase, McLean.
Lieut. H. Grant Preston, Harrisonburg.
Lieut. Chas. W. Warren, Upperville.

Public Health Statistics

I. C. RIGGIN, M.D.,
State Health Commissioner of Virginia.

The report of the Bureau of Communicable Diseases of the State Department of Health for June, 1941, compared with the same month in 1940 and for the period of January through June, 1941, compared with the same period in 1940 follows:

	JUNE 1941	JUNE 1940	JAN.-JUNE 1941	JAN.-JUNE 1940
Typhoid and Paratyphoid Fever	14	13	72	65
Diarrhea and Dysentery	392	96	667	447
Measles	2,825	996	32,570	3,124
Scarlet Fever	47	110	882	1,011
Diphtheria	30	25	214	288
Poliomyelitis	2	2	13	8
Meningitis	8	8	66	46
Undulant Fever	1	2	7	9
Rocky Mountain Spotted Fever	1	6	9	9
Tularemia	0	3	18	28

NUTRITION AND HEALTH

The fundamentals involved in applying the science of nutrition to the human being are of recent origin. Many laboratory discoveries yet are to be substantiated through research work now being conducted in numerous places.

Unlike pellagra, scurvy, beri-beri, and rickets, the signs and symptoms of which are clearly defined and easily recognized, borderline nutritional deficiencies as clinical entities are difficult to establish, assuming that the physician is given an opportunity to make a diagnosis. Most malnourished people rarely consider themselves ill enough to seek medical advice.

Physiologists, chemists and clinical investigators have made available additional information to physicians who consequently are better able now to

handle nutritional problems than ever before. Interest in the subject on the part of the medical profession is becoming more marked. As is well known, discussion of some aspect of nutrition now occurs frequently at medical society meetings; and numerous articles on this subject are to be found in the current medical journals.

Efforts to combat malnutrition both on the part of the private practitioners and public health officials should be directed to all classes and to all ages. Particular emphasis should be placed upon the early development of the child, including that of the mother during the prenatal period. Young women experiencing pregnancy, especially for the first time, seem to be more receptive to health teaching than at any other period. Full advantage should be taken of the opportunities which the prenatal period offers for nutritional instruction.

No matter how much is done about the nutrition of the school child through adequate lunch service at school, so long as candies and soft drinks are readily accessible while at school or through lack of parental discipline, malnutrition will continue to be a problem. The family physician, can continue to emphasize this and other nutritional facts to parents. As is known, children in the higher income group sometimes show the same nutritional disturbances as those in the lower levels. In both cases the cause likely is to be the wrong selection of foods or the use of proper foods at the wrong time.

In common with the practicing physician, public health officials are interested in the prevention of food deficiencies. The general public is inclined to accept nutritional suggestions for hearsay. The present popular trend definitely and generally is towards vitamins, in contrast to the former enthusiasm directed toward calories. The doctor, on the other hand, justifiably is only as rapid in prescribing vitamin concentrates as he feels is compatible with some certainty of results. He knows that in most cases the proper way to approach this problem is through dietary means, and not through the use of super-concentrated drugs and pills. As the medical profession well appreciates, changes in food habits are frequently indicated for many families.

The Virginia State Department of Health contemplates the employment of a nutrition consultant in the near future, thus making specialized assistance available to its nurses. Likewise, the Department's dentists will further stress nutrition in connection with the dental services for school children.

In short, while the problem of nutrition in the mass now is accepted as a public health responsibility, leadership in promoting this subject so far as individual family groups are concerned still rests with the family physician.

Mental Hygiene Activities

During the early part of the year the programs sponsored by the Mental Hygiene Society of Virginia emphasized mental hygiene of adolescence. This emphasis has rather rapidly shifted to the mental hygiene problems associated with national defense. This has been a logical and well worth-while change, for the part that this country should and will take in the present world crisis is uppermost in the minds of all citizens.

The very nature of democracy, with its freedom of thought and the non-restriction of speech, practically precludes a unanimity of opinion, and of course freedom of speech makes it possible for persons to express publicly their discordant views regardless of how illogical or without basis they may be.

Then too there will always be those who are willing to listen to and read about these discordant views largely because they tend to satisfy their "wishful thinking", and they do not stop to analyze these views against the facts.

Under such conditions "National Morale" becomes a topic of extreme importance. The Mental Hygiene Society has rendered a major service to the citizens of Virginia and the country at large by sponsoring a portion of the program of the Institute of Public Affairs, just recently closed at the University of Virginia.

The general theme of this portion of the program was "National Morale", and Drs. Harry Stack Sullivan, Karl Menninger and C. Macfie Campbell and Mr. Edmund Taylor, all men of great knowledge, gave very excellent addresses.

Dr. Sullivan told of the problems associated with the Selective Service for National defense. He pointed out that individuals with neurotic tendencies, personality disorders, or intellectual defects are un-

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able to meet the stresses of army life but that they most certainly have a place in the program of national defense. He stressed particularly that the people of the community should not criticise rejected individuals and emphasized the place of mental hygiene in their proper placement and adjustment.

Dr. Menninger defined civilian morale in this particular crisis as "the maintenance of what we like to regard as the normal spirit of optimism, courage, single-mindedness as to national aims, loyalty to the government, to the cause espoused by the government and to the allies of the government."

Dr. Campbell developed the thought that the National Morale is a composite picture of the morale of its citizenry. He said, "The ability of a nation, as of an individual, to meet a physical or moral emergency will depend upon previous habits of physical and moral exercise. If not in training, the individual exposed to the physical test may soon be out of breath; if his moral faculty has become rather flabby a spiritual challenge may find him inadequate; he may easily become irresolute and discouraged and compromise with his conscience."

Mr. Taylor, who has had very close contact with the German propaganda techniques, disclosed how many American citizens and some newspapers are unwittingly or otherwise, materially assisting Germany to undermine and destroy the morale of this country. The engendering of fear and the development of a state of indecision are the ultimate aims of such propaganda techniques. If our democratic form of government is to survive, and I am sure it is the desire of a great majority that it do so, we must become more unified in the support of our well chosen national leaders. Such unified support will make it much easier for them to do the right thing at the right time.

Indecision is definitely mental disorder and may well develop into frank mental disease. The Mental Hygiene Society of Virginia continues its efforts to reduce the incidence of mental disorder and mental disease.

JOSEPH E. BARRETT.

Book Announcements

Books received for review are promptly acknowledged in this column. In most cases, reviews will be published shortly after the acknowledgement of receipt. However, we assume no obligation in return for the courtesy of those sending us the same.

Start Today. Your Guide To Physical Fitness. By C. WARD CRAMPTON, M.D., Major, Medical Reserve Corps, United States Army; Formerly Director of the Department of Physical Education and Hygiene, New York Board of Education; etc. New York. A. S. Barnes and Company. 1941. xxii-224 pages. Illustrated. Cloth. Price $1.75.

Play For Convalescent Children in Hospitals And At Home. By ANNE MARIE SMITH, Staff Instructor, Leaders' Training School, Community Recreation Service, Chicago, Illinois. A. S. Barnes and Company. 1941. xviii-133 pages. Cloth. Price $1.60.

Infantile Paralysis. A Symposium Delivered at Vanderbilt University, April 1941. Published by The National Foundation for Infantile Paralysis, Incorporated, New York City. Waverly Press, Inc. Baltimore, Md. 1941. vii-239 pages. Cloth.

X-Ray Therapy of Chronic Arthritis. (Including the X-Ray Diagnosis of the Disease). Preliminary Report Based on 100 Patients Treated at Quincy, Illinois. By KARL GOLDHAMER, M. D., Associate Roentgenologist, St. Mary's Hospital and Quincy X-Ray and Radium Laboratories; etc. Radiologic Review Publishing Company, Quincy, Ill. 1941. 131 pages. With 24 original illustrations, two roentgenograms, and four tables. Cloth. Price $2.00.

Essentials of Endocrinology. By ARTHUR GROLLMAN, Ph.D., M.D., Associate Professor of Pharmacology and Experimental Therapeutics in the Medical School of the Johns Hopkins University; Formerly Associate Professor of Physiology and Instructor in Chemistry in the same Institution. Philadelphia. J. B. Lippincott Company. 1941. Octavo of xvi-480 pages. 74 illustrations. Cloth. Price $6.00.

This book stresses the clinical aspects of endocrinology, reviewing briefly the anatomy, histology, physiology, and pharmacology of the endocrines. The author's standing as an investigator in endocrinology is sufficient recommendation for the accuracy of the material, and it will prove well worthwhile to any physician interested in this field. Some interesting inclusions are the structural formulae of the hormones, a brief note concerning the hormones of the gastro-intestinal tract and some presumptive hormones such as the heart hormone. A table of the frequency of appearance of endocrine diseases at the Johns Hopkins hospital over a period of years is included, some of which in their order of frequency are diabetes, hyperthyroidism, hypothyroidism, adrenal insufficiency, adenoma of hypophysis, tetany, pituitary insufficiency, etc. However, one is surprised that out of the large number of cases there was only one case of female hypogonadism. A list of commercial preparations of the endocrines is included on the front and back covers.

However, the sex hormone preparations have not been covered nearly so well or completely as has been done by the late Doctor Crockett (this journal, 67:732-739, 1940).

The reviewer misses under the discussion of ovulation, mention of the peak of gonadotropin excretion which consistently occurs in the normal female, and is believed to have some association with ovulation. This peak might also have been included in the curve illustrating the hormonal interplay in menstruation.

In spite of these minor criticisms, this is an excellent, complete, and up-to-date book.

R. J. M.

The Avitaminoses. The Chemical, Clinical and Pathological Aspects of the Vitamin Deficiency Diseases. By WALTER H. EDDY, Ph. D., Professor of Physiological Chemistry, Teachers College, Columbia University; etc. And GILBERT DALLDORF, M. D., Pathologist to the Grasslands and Northern Westchester Hospitals, Westchester County, N. Y. Second Edition. Baltimore. The Williams and Wilkins Company. 1941. Octavo of xiii-519 pages. Cloth. Price $4.50.

The reviewer considers this book a valuable addition to the vitamin field. It covers the chemical constitution of the known vitamins as well as the clinical and pathological aspects of the vitamin deficiency diseases. It is divided into two parts—*Part I*, The Vitamins and Avitaminoses; *Part II*, Methods of Studying Avitaminoses and The Vitamin Content of Foods.

Each of the vitamins known to play an important role in human nutrition is discussed in considerable detail from five viewpoints: (1) Chemical structure; (2) Nature and function; (3) Clinical and pathological changes in the deficiency state; (4) Requirements; and (5) Distribution in food stuffs. In addition one chapter is devoted to laboratory tests useful in the diagnosis and study of deficiency disease. In this book the authors have presented in a comprehensive but condensed form the enormous literature on vitamins which is of most general interest to the physician and pathologist. The morphological changes noted in various avitaminoses are discussed in detail. Many photomicrographs illustrating the pathological changes noted in avitaminoses are included. The book should prove a most valuable aid to all interested in nutrition or clinical aspects of vitamin deficiencies.

J. C. FORBES.

Methods for Diagnostic Bacteriology. A Complete Guide for the Isolation and Identification of Pathogenic Bacteria for Medical Bacteriology Laboratories. By ISABELLE G. SCHAUB, A.B., Assistant in Bacteriology, Department of Pathology and Bacteriology, The Johns Hopkins University School of Medicine. And M. KATHLEEN FOLEY, A.B., Bacteriologist in Charge of the Diagnostic Bacteriological Laboratory of the Medical Clinic, The Johns Hopkins Hospital. St. Louis. The C. V. Mosby Company. 1940. 313 pages. Cloth. Price, $3.00.

This book will find a useful place in the field of diagnostic bacteriology. The number of standard textbooks available more than adequately cover the fundamental and theoretical aspects of bacteriology. The hospital bacteriology laboratory is called upon to carry out a variety of examinations and the importance of rapid, accurate diagnosis need not be elaborated upon. This test incorporates as one of the authors states "the tricks of the trade"—essential for carrying out these procedures.

Not all of the procedures will find equal use in other laboratories, but this volume is an admirable start toward a standard procedure for diagnostic laboratories.

J. DOUGLAS REID.

An Introduction To Dermatology. By RICHARD L. SUTTON, M.D., Sc.D., LL.D., F.R.S. (Edin), Emeritus Professor of Dermatology, University of Kansas School of Medicine. And RICHARD L. SUTTON, JR., A.M., M.D., L.R.C.P. (Edin), Assistant Professor of Dermatology, University of Kansas School of Medicine. Fourth Edition. St. Louis. The C. V. Mosby Company. 1941. Octavo of 904 pages. With 723 illustrations. Cloth. Price $9.00.

The following statement is taken from the preface of this edition, "This book is intended for students, collegiate and postgraduate, contains a review of dermatology sufficient to outline the scope of the specialty beyond provincial boundaries. It contains considerably more than a student may encompass, but he can read less of it."

This edition is a condensed volume of the larger tenth edition, plus a review of the important dermatological literature since 1939. All descriptions are clear and concise. There is a well selected bibliography. The book is well illustrated and it is printed on excellent paper. It may be highly recommended to students, general practitioners, and those limiting their practices to dermatology and syphilology.

ALLEN PEPPLE, M.D.

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Editorial

The Internship.

It will not be surprising to those who are aware of the rapid changes through which medicine is passing to discover that the internship is very different from what it used to be.

In the early days the young Lydgate was a "house doctor", a "resident physician" or a "house pupil". Not until after the Civil War did the term "intern" come into common use. The old internship of two years, with six months in the laboratory, six months as a junior intern, six months in the specialties, and six months as a senior house officer, presumably fitted a man for anything. If he was lucky enough to serve in a large municipal hospital he saw a riot of diseases, chiefly in the advanced stage, and went out rather cocky about what he could do. It was natural for him to feel and act that way, because many of his fellow graduates went straight into practice without any such experience. The man with the good internship behind him perhaps had a right to feel his superiority.

Times have changed. The internship as viewed today has two objectives: (1) the rounding out and practical application of a medical school course, and (2) the training of a man to accept responsibility. The length, content and character of the internship today depends upon this concept. It is really a continuation of the clinical clerkship with added responsibility. It is the time for continued fundamental work in medicine, the time to develop life habits of study and permanent professional attitudes. The modern internship is designed to fit a man for general practice, or for the residency which in turn leads to specialization. There are today several recognized categories of the internship.

There is first the rotating internship in which a man rapidly in the course of a single year passes through medicine, surgery and the various specialties. In the United States more than 6,000 young doctors enter such internships each year. They constitute two-thirds of the total number of positions available. There is, however, the danger that this type of internship may be carried to an extreme with rotation occurring as often as every three weeks, affording little more than a nodding acquaintance with the specialties concerned. There are still a few straight internships, about 500 of them. They are regarded as a proper intermediary between the rotating internship and the residency. There are also mixed internships. There is now a new type under discussion known as the "case internship", in which a man follows one case through the outpatient department and the various services of the hospital as often as that patient is admitted to the hospital during his term of service. He even visits such patients in their homes. The type of internship,

however, which is looked upon with greatest favor has been called the "general internship". It is based upon the principle that the intern should spend most of his time in general medicine, pediatrics, obstetrics, surgical diagnosis, minor surgery and first aid. The day is considered past when an intern should spend his time learning the special techniques of the specialties. It is believed that he learns most about the specialties by consultation with specialists in the general wards of the hospital.

Ideally the modern intern should be taught to view the patient as a whole, his mental as well as his physical state, his environment and his reaction to it, as well as the pathologic changes responsible for symptoms and signs. For this sort of instruction, the clinical professor who does not live the cloistered life, who rubs elbows with a variety of sick people, visits them in their homes, takes time to talk over their woes with them in his office, is best fitted.

The intern's assignment to the outpatient department offsets the one-sidedness of hospital practice where only disease in its advanced stage is encountered. In the outpatient department the intern develops an awareness of disease in its incipiency and in its more chronic manifestations and learns neither to fear, to scorn nor to neglect the victims of life long disability.

The hospital occupies the key position in the education of medical student, intern and resident. Its obligation to provide a program with an adequate educational content for interns is unquestioned. Unless such a purpose is systematically and enthusiastically carried out the internship is not worth much. To develop such a program properly the hospital should employ a qualified man to act in the capacity of Director of Intern Education. On the other hand the medical school's responsibility does not cease with the graduation of its students. It should see that the holders of its degrees secure internships of the proper character, and it should cooperate with neighboring hospitals (many of them not connected with a medical college) in the establishment of adequate educational programs for their interns.

In the modern hospital the intern no longer rides the ambulance, administers anaesthetics, serves as admitting officer, or performs routine laboratory work. The hospital should employ salaried men for these chores and should not waste the valuable time of interns on them.

An intern should have responsibilities but he should not be left to his own devices without supervision by residents and staff officers. He should be busy but not overworked. A case load of eight to ten patients, or the admission of a patient a day, is enough for any man. By the time he has taken a history and made a physical examination, done the special laboratory work on the patients in which he is interested, attended an autopsy or two, supervised medical students, made routine ward rounds, taken care of the daily emergencies to which he is summoned, attended organized ward rounds by his chiefs, listened in on one or two consultations with specialists, attended a clinical, a clinicopathologic, clinicophysiologic, radiologic, or medicopsychiatric conference, a staff meeting or a journal club, looked up some subject in the Index Medicus, thumbed through the latest medical journals, read several articles to which he is attracted, kept his daily log, written the reports required of him, talked with his staff adviser and perhaps been in conference with the Educational Director of Interns, he deserves a game of ping pong or an hour on the handball court, and sometimes an afternoon off for golf. If his hobby is the humanities or some other cultural activity he should now and then have an opportunity to find recreation in them.

The American Medical Association Recognizes the General Practitioner.

At the last meeting of the American Medical Association the House of Delegates voted the creation of a new section on General Practice. Although there had been for a long time agitation for such a section, the move came with unexpected suddenness. It is perhaps a good thing that general practitioners attending the meetings of the American Medical Association in the future will find a section in which they can be at home, untrammeled by the "highbrows" and the technical jargon of specialists and full-timers. Perhaps it is another step in the direction of the setting-up of another category of certification, and the time may soon be expected when on the walls of the general practitioner's office will hang a diploma certifying to the fact that he is not an obstetrician, not a gynecologist, neurologist, psychiatrist, pediatrician, cardiologist, gastro-enterologist, syphilologist, allergist, dermatologist, roentgenologist, or otorhinolaryngologist, or even an internist, but just a doctor, a darn good one you can be sure.

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Warning.

No part of William L. Shirer's *Berlin Diary* (New York, Alfred Knopf, 1941) will be more arresting to medical men than the two entries, *Berlin, September 21, 1940,* and *Berlin, November 25, 1940,* relating to the "mercy killings" of the insane that are believed to have taken place in the Reich with administrative sanction, some say to the number of 100,000. In explanation it is suggested that they are carried out to save food, to experiment with new poison gases and death rays, to forward the sociologic and eugenic ideas of Nazi extremists, to release able bodied Germans caring for institutional cases (one for every three or four patients), and to afford hospital space for war wounded. Whatever the reasons for them, if these murders are being committed as Shirer believes, it must be concluded in his words: "It's a Nazi, messy business". It will be abhorrent to Medicine as to the Church that has already protested. The Vatican on December 6, 1940, stated that "the killing of those who, although they have committed no crime worthy of death, nevertheless are considered no longer useful to society or the state because of physical or mental deficiencies, is contrary to both natural and divine law".

While some may doubt Shirer's story of horror, none may doubt that uncontrolled power in the hands of one man or party could make such a horror possible. American medicine will find in the accomplished fact or imagined fiction one more cogent reason to bend its efforts, individual and corporate, to forbid an entry of dictatorship upon the shores of America and its own sacred domain.

Department of Clinical and Medical Education of the Medical Society of Virginia

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Summer Short Course.

The second summer short course in medicine was held at the University of Virginia during the week of June 16-21, 1941. Twenty-eight members of the faculty of the Department of Medicine took part in the instruction. In addition Dr. Warfield M. Firor, Associate Professor of Surgery, Johns Hopkins Medical School, and Dr. Walter O. Klingman, Associate in Neurology, College of Physicians and Surgeons, were on the program. The morning sessions were devoted to lectures while the afternoon was given over to clinics and ward rounds. Evening meetings consisted of conferences, lectures, round-table discussions and a banquet.

The following doctors attended the course:

Dr. S. K. Ames, Cape Charles.
Dr. F. L. Byers, Harrisonburg.
Dr. Glenn C. Campbell, Staunton.
Dr. J. G. Cox, Hillsville.
Dr. A. B. Grubb, Cripple Creek.
Dr. Percy Harris, Scottsville.
Dr. C. W. Hickam, Pulaski.
Dr. H. G. Hudnall, Covington.
Dr. M. B. Jarman, Hot Springs.
Dr. N. B. Jeter, Covington.
Dr. Joseph H. Low.
Dr. J. C. McCluer, Alexandria.
Dr. H. C. McCoy, Gordonsville.
Dr. A. M. McLaughlin, Waynesboro.
Dr. Joseph L. Mann, Hampton.
Dr. E. R. Moorman, Kilmarnock.
Dr. W. A. Murphy, Staunton.
Dr. E. F. Neal, Altavista.
Dr. Dan O. Nichols, Charlottesville.
Dr. W. R. Pretlow, Warrenton.
Dr. V. W. Quillen, Nickelsville.
Dr. A. C. Ray, Jr., Ashland.
Dr. A. C. Ray, Sr., Lynnhaven.
Dr. Paul Revercomb, Charleston, W. Va.
Dr. John E. Roberts.
Dr. L. G. Roberts, Moormans River.
Dr. Alex Robertson, Staunton.
Dr. E. B. Robertson, Danville.
Dr. Elizabeth Sherman, Front Royal.
Dr. Frank E. Tappan, Berryville.
Dr. George A. Torrence, Hot Springs.
Dr. W. R. Warren, Woodberry Forest.
Dr. W. R. Watkins, South Boston.
Dr. B. K. Weems, Waynesboro.
Dr. M. J. W. White, Luray.
Dr. George C. Williams, Jewell Ridge.
Dr. Thomas V. Williams.

Local Short Course.

As previously announced, a short postgraduate course in Internal Medicine and Surgery was held at Leesburg at the request of the Loudoun County Medical Society during April and May. Speakers on the program came from the University of Virginia, Medical College of Virginia, and Duke University. Thirteen doctors from Loudoun County attended the course.

GEORGE B. ZEHMER,
Executive Secretary.

Proceedings of Societies

Warwick County Medical Society.

At the annual meeting of this Society, Dr. O. C. Jones was elected president, Dr. H. W. Potter, vice-president, and Dr. Paul Hogg, secretary-treasurer. All are of Newport News.

The Fauquier County Medical Society,

At its meeting on May 28, elected the following officers: President, Dr. Henry L. Townsend, Marshall; vice-presidents, Drs. V. L. McCullers, Remington, and George H. Davis, Warrenton; and secretary-treasurer, Dr. William R. Pretlow, Warrenton (re-elected). Delegate and alternate to the State meeting were also named at this time.

The Lynchburg Academy of Medicine

Held its regular meeting on June 2 with Dr. Powell Dillard, president, presiding. Two local dentists, Drs. Clarence Garrard and Harry Shotwell, presented papers on "Factors Influencing Facial Form" and "Dental Infection and X-Ray Interpretation", which were thoroughly enjoyed by the members present.

C. E. KEEFER, *Secretary.*

News Notes

The State Meeting.

Plans are well under way for a meeting of outstanding interest at Virginia Beach, October 6, 7 and 8. The business sessions—Council and House of Delegates—will be held on Monday 6, so as to complete as much business as possible before the scientific sessions which are to commence on Tuesday morning. The Tuesday morning session will be a general one and will include an address by the President, papers by several members, and a pathological conference by Dr. Lewis Hamman of Baltimore. That afternoon, Colonel Norman Kirk, M. C., commanding officer of the Walter Reed Hospital, Washington, will be the guest speaker, and, following his address, there will be two sections for papers by members. The same plan will be adopted on Wednesday morning, with a gathering about noon to hear Dr. James R. Miller of Hartford, who will speak on an obstetrical subject. Dr. Henry Cave of New York will give a surgical address in the afternoon, following which there are to be four panel discussions—General Practice of Medicine, Surgery in Relation to General Practice, Pediatrics, and Obstetrics and Gynecology. These are to replace the Round Tables and it is believed will prove of much interest.

Tuesday evening will be free for alumni and special group dinners and meetings, and on Wednesday, a banquet with floor show and dancing will close the 1941 meeting.

Scientific and commercial exhibits, a hobby display, golf and tennis tournaments, swimming in the Cavalier in-door pool, and other amusements will add to the pleasure of those attending.

Make your reservations and plan to attend this meeting.

Tennis Tournament.

Plans are being made for a tennis tournament to be held during the meeting of the State Society. Matches will be played on Sunday, October 5, and on Monday, October 6, and a prize will be awarded.

Members interested in playing tennis are requested to notify the Society's office or Dr. B. E. Harrell, Medical Arts Building, Norfolk, Virginia, at least ten days before the meeting.

Dr. Wiley Davidson Lewis,

Formerly assistant superintendent of the Western

State Hospital, Tennessee, has joined the staff of Saint Albans Sanatorium in Radford. He is a graduate of the University of Tennessee College of Medicine and has recently completed a Fellowship in Neuropsychiatry at Adams House, Boston.

Dr. Allen W. Lane

Is located in Blackstone where he is engaged in general practice. He recently resigned as health officer of Pulaski County.

Dr. Alex. N. Chaffin,

Formerly at Catawba Sanatorium, is now with the Clinchfield Coal Company Hospital at Dante.

Dr. James H. Gressette,

Of the Staff of the Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, has returned from Philadelphia after taking a special course in bronchoscopy and laryngeal surgery at the Graduate Hospital of the University of Pennsylvania.

Dr. T. J. Humphries,

Formerly of Culpeper, is now associated with Dr. E. Berkeley Neal in the practice of pediatrics, with offices in the Shenandoah Life Building, Roanoke. Dr. Humphries is a graduate of the Department of Medicine, University of Virginia, in 1938 and has been recently connected with its department of pediatrics.

Dr. S. A. Draper,

Formerly of Roanoke, is now located at 122 North Thornton Street, Orlando, Fla.

Dr. Herbert Carl Lee

Announces the opening of his offices in the Medical College of Virginia Hospital, his practice being limited to surgery. He has recently completed a residency in surgery at the College.

Dr. E. W. Perkins,

Richmond, was certified a Diplomate of the American Board of Ophthalmology at a recent meeting of the examiners in New York City.

Dr. Reuben F. Simms,

Richmond, was recently elected a vice-president of the Lions Club.

Dr. F. N. Mullen, Jr.,

Announces that he is now located at 203 Medical Arts Building, Norfolk. He has recently been at South Mill, N. C.

Dr. Lyddane Miller,

Formerly of Piney River, is now located for practice at Amherst Court House.

Dr. Louise Fry Galvin

Of Richmond has been appointed to the staff of the State Department of Health as pediatric consultant of the orthopedic division of the Crippled Children's Bureau and medical supervisor of the Department's Children's Rheumatic Fever Program.

Dr. Galvin is assistant professor of pediatrics at the Medical College of Virginia and has been practicing her speciality during the last six years in Richmond. She formerly was medical director of the Methodist Orphanage and of the Belle Bryan Day Nursery.

Southern Medical Association Announces Change in Days of Meeting.

The Southern Medical Association meeting will be held in St. Louis on Monday afternoon, Tuesday, Wednesday and Thursday, November 10-11-12-13, instead of Tuesday, Wednesday, Thursday and Friday forenoon, November 11-12-13-14, as previously announced.

The Association will open at noon on Monday, the registration beginning at that time, the scientific programs beginning at 2:00 P. M. and continuing through Tuesday, Wednesday and Thursday, all Association activities being concluded in the late afternoon of Thursday. The registration, scientific and technical exhibits, all clinical sessions, all sections and all conjoint meetings, will be held at the Municipal Auditorium.

The general session, open to the public, will be held on Monday evening; the general session for the address of welcome, the president's address and the report of council, followed by the president's reception and ball, will be on Tuesday evening; and the alumni reunion dinners will be on Wednesday evening, all these evening activities to be held at the Jefferson Hotel.

Dr. Coleman Resigns From University of Virginia Professorship.

Dr. Claud C. Coleman, who since 1937 has had the unique distinction of occupying two professorial chairs at the same time, one at the University of Virginia and one at the Medical College of Virginia, has resigned as Clinical Professor of Neurological Surgery in the Medical Department of the University of Virginia, effective August 1, 1941.

Dr. J. M. Meredith, who was Assistant Clinical Professor under Dr. Coleman, has also resigned. He becomes Associate Professor of Neurological Surgery at the Medical College of Virginia, and in the future will be associated with Dr. Coleman in his private work.

Dr. Crutchfield Assumes Professorship at the University of Virginia.

Dr. W. Gayle Crutchfield, who since 1937 has been Associate Clinical Professor of Neurological Surgery at the University of Virginia, has recently been promoted to the head of the department, succeeding Dr. C. C. Coleman, resigned. He will shortly move to Charlottesville to assume this new position. He will be missed in Richmond where he has held an assistant professorship in the Medical College of Virginia and been associated with Dr. Coleman in private work.

Dr. A. M. Showalter Elected Officer of Rotary International.

Elected an officer of Rotary International at the international convention held in Denver, Colorado, in June, Dr. A. M. Showalter, president and chief surgeon of the New Altamont Hospital in Christiansburg, took office on July 1 as a district governor of Rotary International. Unanimously elected by convention delegates representing the majority of the 212,000 Rotarians and 5,000 Rotary clubs of more than sixty countries of the world, Dr. Showalter will devote much of his time during the year to visiting each of the forty-four Rotary clubs of western Virginia which comprise his district.

News From the Medical College of Virginia.

Faculty promotions for the fiscal year beginning July 1, 1941, are as follows.

Dr. Webster P. Barnes, from associate in to assistant professor of surgery.

Dr. Guy W. Horsley, from associate in to assistant professor of surgery.

Dr. Lawther J. Whitehead, from assistant professor to associate professor of radiology.

Dr. Thomas D. Rowe, from assistant professor to associate professor of pharmacy.

Dr. Rudolf Thomason, from associate in to assistant professor of ophthalmology.

Dr. Delbert A. Russell, from assistant to instructor in radiology.

Dr. Edward A. Delarue, Jr., from assistant to instructor in medicine.

Dr. John P. Lynch, Jr., from assistant to instructor in medicine.

Dr. W. Hughes Evans, from instructor to associate in obstetrics.

Dr. W. C. Winn, from instructor to associate in obstetrics.

Dr. Walter J. Rein, from assistant to instructor in ophthalmology.

Dr. William A. Johns, from instructor to associate in surgery.

Dr. Robert V. Terrell, from instructor to associate in proctology.

Dr. E. I. Evans, from assistant to instructor in surgery.

Dr. John Robert Massie, from assistant to instructor in surgery.

Dr. Charles M. Nelson, from instructor to associate in urology.

Miss Edna J. Townsend, from instructor to associate in pediatric nursing.

Miss Mabel Blount, from assistant to instructor in dietetics.

Miss Harriet Stevens, from assistant to instructor in dietetics.

Dr. Allen Pepple, from assistant to instructor in dermatology and syphilology.

Dr. St. George Tucker, from assistant to instructor in medicine.

Miss Ann Parsons and Miss Edna Townsend, who have been on leave of absence for study returned to the school of nursing on July 1.

Dr. William T. Sanger, president, attended the Institute for Officials of Higher Institutions at the University of Chicago the second week in July.

The psychiatric floor of the new hospital opened July 21. Thirty-eight beds will be available.

The Association of American Medical Colleges has accepted the invitation of the college to hold its annual meeting here, October 27, 28 and 29.

Gifts and grants to the college for the fiscal year ending June 30, 1941, totaled $366,844.34.

Southern Tuberculosis Conference Meets at Asheville.

Subjects to be discussed in the medical section of the Southern Tuberculosis Conference at Asheville, N. C., September 15, 16, and 17, include: Laryngeal Tuberculosis; Collapse Therapy in the Negro; Tuberculosis of Bone and Joints; Silicosis;

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Relation of Tuberculosis to National Defense; False Security in an Apparently Effective Pneumothorax; Methods of Procedure following Ineffective Pneumothorax; Extra-Pleural Pneumothorax and Thoracoplasty; Isolation of the Open Case; Vitamins and Tuberculosis; Tuberculosis in Industry; Tuberculosis in the Aged; and Tuberculosis in Mental Hospitals.

A number of distinguished speakers have been secured to present the papers on this program, many of them being physicians of national reputation.

Dr. Joseph R. Blalock, Superintendent of Southwestern State Hospital, Marion, will present the paper on tuberculosis in mental hospitals.

The complete program of the conference may be had by writing the Virginia Tuberculosis Association, 504 Atlantic Life Building, Richmond. It is expected this will be ready for distribution about the first of August.

Married.

Dr. John Washington Clark, Front Royal, and Miss Sallie McArthur Mason, Ridgeway, June 24. Dr. Clark is a graduate of the Medical College of Virginia, class of '38.

Dr. Stanley H. Macht of Crewe and Miss Naomi Newman, daughter of Dr. and Mrs. Samuel Newman of Danville, July 1.

Dr. Alva Duckett Daughton, East Falls Church, and Miss Vera Alice Hanson, Richmond, July 18.

Dr. H. B. Webb,

Waynesboro, has recently been named physician in charge of the Community Hospital, in which capacity he will act as administrator as well as continue his practice.

X-Ray Unit For Sale—

Complete radiographic and fluoroscopic outfit, including all darkroom equipment. Reasonable. Owner called in military service. Address "X-Ray", care VIRGINIA MEDICAL MONTHLY, 1200 East Clay Street, Richmond. (*Adv.*)

Wanted—

Medical microscope in good condition for study work. Write "Student", care the MONTHLY, 1200 East Clay Street, Richmond. (*Adv.*)

For Sale

Short wave diatherm. In first-class condition. Reasonable price. Apply No. 175, care this journal. (*Adv.*)

Doctor's Office for Rent.

One or two room suite in Richmond, ideally located for specialist or general practitioner. Joint reception room and laboratory with established doctors. Address "Office", care this journal, 1200 East Clay Street, Richmond. (*Adv.*)

Obituary Record

Major Allen J. Black,

Medical Corps, U. S. Army, retired, died as a result of a heart attack at his home in Richmond June 25. He was a native of Salem and seventy-six years of age. Dr. Black graduated from the Medical College of Virginia in 1884, following which he practiced in Radford and Roanoke before entering the medical service of the army. He saw service in Cuba during the Spanish-American War, in the Philippines during the Insurrection, and in the World War, and was retired in 1928 because of physical disability. Dr. Black had been an interested member of the Medical Society of Virginia since 1884 and he was president of the Roanoke Academy of Medicine in 1909. His wife survives him.

Dr. Joseph Nicholas Applewhite,

Capron, died May 5. He was seventy-four years of age and a graduate of the Medical College of Virginia in 1895. Dr. Applewhite had been a member of the Medical Society of Virginia for thirty-seven years.

Dr. Richard Franklin Slaughter, Jr.

Augusta, Ga., died July 3 after an illness of six months. He was a native of Hampton, Va., and was thirty-five years of age. Dr. Slaughter practiced for a while at Norfolk but for the past four years has been in Augusta where he was head of the department of neuro-surgery of the University of Georgia Medical School. He was formerly a member of the Medical Society of Virginia.

Dr. Edgar A. Pole,

Who had practiced at Hot Springs for thirty-five years, died July 19 after a short illness. He was seventy-one years of age and a graduate of the former Baltimore Medical College in 1897. Dr. Pole had been a member of the Medical Society of Virginia for forty-three years. His wife and five children survive him.